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OBSCURE LESIONS OF THE SHOULDER; RUPTURE OF THE SUPRASPINATUS TENDON

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THE chief point of this paper is to advocate early exploratory incision in cases of severe shoulder injuries with negative X-Rays.

I wish I had the best of reasons for defending this position—namely that I had repeatedly done what I advise and been glad that I had. As a matter of fact I have never done an exploratory operation on an early case of injury to the shoulder nor do I remember ever having the opportunity. Nor am I likely to see such cases in the future, for I have no connection with any clinic which receives accident cases. However, many of you men are seeing and treating acute traumatic shoulder lesions. I do see many chronic cases because for about ten years, thanks to Dr. Francis Donoghue's policy of sending cases for impartial examination for the Industrial Accident Board of Massachusetts to those who have given study to particular classes of work, I have seen many cases of long standing shoulder injuries. Also for a number of years some of our insurance companies have asked me to operate on their patients with obscure lesions in this region. This has formed the bulk of my experience on which to base my arguments about early incision. I realize that seeing these selected difficult cases gives me a biased point of view, for I do not see the perhaps large number of cases which are satisfactorily and promptly treated. My ideas of routine work comes from 15 years of experience in the outpatient department of the Massachusetts General Hospital when I intensively studied a series of 100 shoulder cases and wrote my papers on subacromial bursitis, a bibliography of which appears at the end of this article.

Speaking from this point of view, therefore, it seems to me that shoulder injuries which are not obvious dislocations or fractures receive scant attention from the surgeons of New England.

Ruptures of the supraspinatus tendon especially are seldom recognized and promptly and efficiently treated. It is to this particular, and I believe fairly common lesion that I wish

to call your attention, for it can be diagnosed, the diagnosis confirmed by exploratory incision, and a fairly satisfactory repair made even in long standing cases. Therefore I believe that some of you who have opportunity, may recognize this very disabling lesion early and perform a really easy and satisfactory operation, promptly.

You probably call these cases, following my own lead in 1908, instances of subacromial or subdeltoid bursitis. If you will reread my papers you will find that even 18 years ago, I attributed most cases of traumatic bursitis to ruptures of the tendon. I am much more inclined to this opinion now. Roughly one-third of all shoulder cases referred to me I believe to be instances of this lesion, although I have operated upon only a small percentage of them.

Any surgeon who may be interested in my proposition of early exploration in shoulder injuries, will find it much to his advantage to dissect this region on a cadaver to learn the relations of the subacromial or subdeltoid bursa to the tendons of the supraspinatus and long head of the biceps. One cannot satisfactorily repair these tendons without knowing the normal relations. The accompanying illustrations do not take the place of actual dissection of the regional anatomy. A fresh cadaver at autopsy is much more satisfactory material than the stained subjects of the dissecting room.

Assuming that I am right that early exploration might save many months of disability to some of these patients and many dollars to the Insurance Companies, what are the opposing arguments—the dangers, the complications, the obstacles, the forms of failure, the pains, discomforts and after effects of these operations?

Take the simple exploratory incision first. i. e., an inch and a half incision through the deltoid, opening the bursa and rotating the arm to inspect the base of the bursa. I believe this to be as absolutely harmless as any minor surgical operation. I have never had, nor can I conceive of any serious complication resulting from

it, even if infection should occur, unless there existed some lesion in the bursa before the operation. Even if infected, the bursa would drain toward the surface and would not involve the joint, for the base is separated by a good one-quarter inch of tendon from the joint as well as by the capsule and two synovial surfaces.

A negative exploration therefore, is negligible except for slight discomfort to the patient and some small expense to the Insurance Company. With increasing experience on the part of the surgeon negative explorations would be rare. In only two cases have I made a negative exploration, where I had diagnosed a rupture of the tendon of the supraspinatus in long-standing cases.

Suppose that the exploration is positive when the bursa is opened and one looks down directly on the cartilage of the joint through the gap in the torn tendon, what then of dangers and complications?

Of course we are confronted with the general dangers of joint surgery, hemorrhage or sepsis resulting in restriction. Years ago I opened a septic shoulder joint with a result of ankylosis in the position of election. The woman had a very useful painless arm—one which I should prefer to the ordinary result in unoperated complete rupture of the supraspinatus. This has been the only case of ankylosis to occur in my practice, and even if such a condition occasionally resulted the patient would hardly be worse off.

The "sabre-cut" incision is of course a more serious operation and the dangers of diastasis of the acromio-clavicular joint and non-union of the acromion, are added to those of sepsis and hemorrhage. I do not advocate its general use unless it is impossible to suture the tendon through the exploratory incision. The chief objection to it is not so much the danger of it, but the long period one must wait for union of the acromion to occur—i. e., from three to four weeks. If one does it at all, the tendons should be freed backward into the scar sufficiently to mobilize the muscle which is more or less elastic. As in other instances in surgery perhaps the thorough operation is the really conservative one. I think it is best to allow the parts to heal in adduction, i. e., the sling position, although I have also succeeded in adduction. This is far less comfortable to the patient however and more trying to the surgeon in doing dressings. I have had no bad results even from the sabre-cut incision, but in a few cases the amount of increased function has been so little that it has hardly repaid the patients for their pain and soreness. However the problem in a recently ruptured case would be much easier for all concerned.

I do not pretend that in the 20 or 30 cases of complete rupture of the supraspinatus that I

have operated on that all has gone smoothly, or entirely satisfactorily. I still regard the operation as difficult and the convalescence as trying and the results only good, not perfect. What I do say is that if I can get fairly good functional arms in cases averaging a year after the injury, you men who are on active duty at hospitals receiving acute traumatic cases can get perfect results if you operate promptly on these cases. You will have no difficulty in recognizing the anatomical structures within a few weeks after the injury but with each month that passes they become more retracted, more fixed, harder to recognize and to repair, and slower in regaining function.

My greatest difficulty with these old cases has been in controlling the discharge of synovial fluid after the operation. Owing to the prolonged irritation of the joint there is an excessive amount of fluid, practically a chronic synovitis such as we find in water on the knee. After the operation this fluid soaks into the surrounding areolar tissue causing redness and swelling and often an acute febrile condition as if the patient reacted badly to a large dose of his own serum. At this stage the wound appears septic and in my early cases caused me much worry, but I have found it unwise to open them to let the fluid out. I think it is better to wait for it to discharge itself. When it does so it is of a clear amber character like hydrocele fluid. It may run for days or even weeks. Sometimes, however, nature takes care of it and the wound does not open.

It is the punctilious care of the dressings in these cases, which makes the convalescence trying. If the coöperation of the patient is obtained I keep the arm in abduction. If not, in any position the patient can make it most comfortable. After the operation it is weeks or months before good function is obtained in these long standing cases, but I believe that in fresh cases the convalescence would be even more brief than in knee joint surgery. I think we should not find this tendency to seepage of joint fluid.

The operation itself where the tendon is much retracted is difficult in some cases unless the "sabre-cut incision" is used. I have special short strong needles which I can use under the acromion. I sometimes use silk but more often catgut. Sometimes when the tendon has torn out from the tuberosity leaving no stub. I drill holes in the tuberosity and pass a mattress suture through. Even if only a suture "a distance" is made, new tendon will form along it.

My results have all been more or less successful, but seldom prompt and perfect. I think all patients have been pleased in the end, but impatient at the slow return to function.

Let me say that there are all degrees of rupture, from a little nick in the tendon to complete evulsion of the supraspinatus

and the adjoining parts of the infraspinatus and subscapularis. The biceps tendon is sometimes torn too. The mild ruptures repair themselves but they are slow. The complete ones remain permanently disabled and suffer more than the average fracture case.

I make no claim that my technique is fully developed and that these cases can be repaired as easily as an inguinal hernia. The comparison with loose semilunar cartilages in the knee is more exact; when the operation for ruptured supraspinatus has been done as frequently, the technique will be as well established. For eighteen years I have been a pioneer in this operation, doing occasional cases. I have published nothing on it since the first two cases, because I have not felt that I had standardized the operation and too many surgeons who "try anything once" would undertake it without due study of the anatomy. However Industrial Surgery now demands the return of these patients to work and they are bound to be experimented on, so I contribute my bit of experience and say "do it early." Exploration is harmless. Make a few mistakes in negative explorations if necessary but you will not delay convalescence if mistaken and you will greatly shorten it if you make the right diagnosis.

If a patient has a severe wrench of the shoulder, and within a day or two can endure passive abduction, but cannot perform active abduction, and the X-Ray is negative, I believe exploration should be done.

If you and the patient cannot make up your minds immediately after the injury and find three weeks later that there is no improvement, consider the question of exploration again. A jog in the motion of passive abduction and a velvety crepitus over a tender point beneath the acromion anteriorly, add greater certainty to the diagnosis. Some day we shall inject air or non-radiable fluid into the bursa and diagnose these cases easily.

Please remember that I do not advocate incision in every injured shoulder, but when a strong laboring man after a severe fall or wrench alleges that he cannot raise his arm, cannot sleep on account of pain, refuses to work, lets his wife dress him—take him seriously, even if the X-Ray is negative and you can detect nothing on physical examination. He may have one of these obscure lesions in which the prognosis is worse than in an out-and-out fracture of the surgical neck of the humerus. Do not be misled by the comparative mildness of his symptoms. If he cannot actively abduct his arm, and especially, if, after you have abducted it for him, he cannot hold it at the horizontal against moderate downward pressure, you can be sure enough of the diagnosis to justify exploratory incision of the bursa. Let me warn you however that the patient must make an honest ef-

fort at abduction as shown by the feeling of contraction of the deltoid muscle beneath your hand, for any soreness about the shoulder is apt to inhibit contraction of the deltoid in a sensitive patient.

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EXPLANATION OF ILLUSTRATIONS

BY MR. H. F. AITKEN

(From drawings made from dissections by the author)

FIGURE 1

The Exploratory Incision to Be Used in Acute Traumatic Cases



The incision starts near the tip of the acromion close to the acromio-clavicular joint and is carried downward for not more than two inches. This will not divide the anterior fibres of the circumflex nerve which might be injured by a longer cut. Beneath the separated fibres of the deltoid muscle is the roof of the bursa and this is picked up and incised as one incises the peritoneum in doing a laparotomy. This "roof" or upper layer of the bursa is depicted as held apart by sutures. Normally this layer is extremely thin like the peritoneum and is so transparent that before incising it, if one holds the wound open with retractors while an assistant rotates the arm, the base of the bursa can be seen rotating beneath it. After the bursa has been incised we see as in this diagram, a smooth, white, globular, convex surface resembling joint cartilage. One would almost think it the articular surface of the humerus turned outward. In reality it is the base of the bursa which covers and adheres tightly to the two tuberosities and the tendinous portions of the supra- and infra-spinatus and subscapularis. However, these structures are not distinguishable on looking into a normal bursa, for they are all welded into one uniform convex surface which rotates as easily as an eye-ball and passes in and out under the acromion as the assistant moves the arm about. One even sees no depression between the two tuberosities where the bicipital groove lies, although with care this can be palpated.

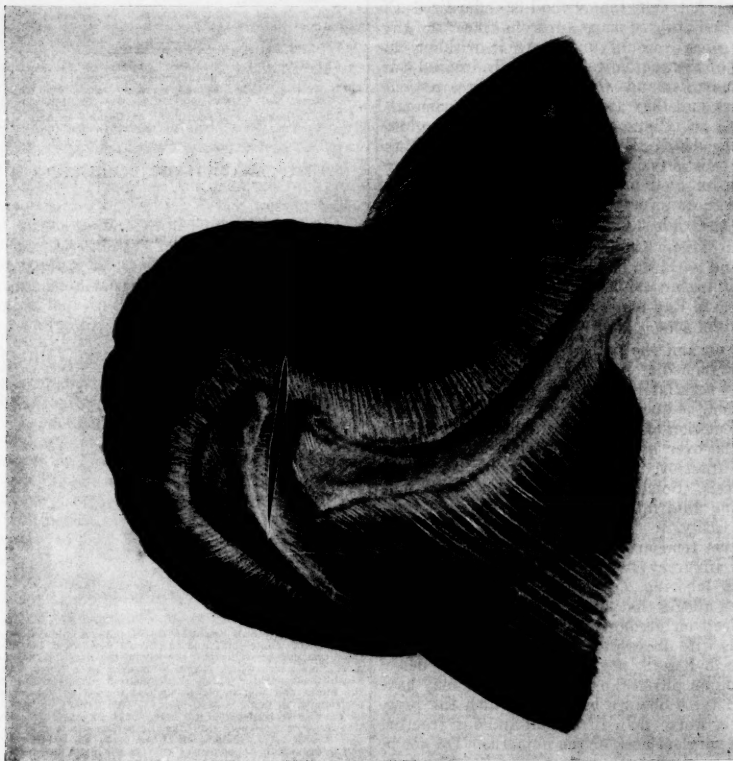
All one sees in the normal case is a smooth white surface and an occasional plicating fold, one of which is depicted in the diagram as a curved line crossing the bursa. These folds constitute movable partitions in, or peripheral extensions of, the bursa. Being excessively thin their two layers can reduplicate on one another or stretch out into a single sheet in any extreme position of the joint. The area of convex surface of the base of the bursa is approximately that of a silver dollar. The lower hemisphere has the bone of the tuberosities beneath it; the upper hemisphere has the tendons of the short rotators beneath it. On inspecting the bursa as in this diagram you are therefore separated from the true joint by a good quarter inch of strong tendinous substance as well as by the capsule and two synovial layers. However, these structures are in fact so closely incorporated together that they rupture as one. I assert this from observation made only in long-standing cases, for I have had

no opportunity to operate immediately after the injury. In fresh cases I should expect to find it very easy to recognize exactly each of the torn tissues. This is difficult in long-standing cases, owing to retraction of the proximal ends.

I speak of this incision as the routine bursal incision, and consider it a practically harmless minor operation. It can usually be made with local anaesthesia only. It is adequate for excising inflamed plicating folds, fringes and bands in chronic cases, or for removing calcified deposits beneath the base of the bursa. The latter appear on exploration as whitish eminences surrounded by red injected areas of inflamed tissue. They resemble boils and when nicked with the knife exude whitish ointment-like or yellowish gritty material evidently under pressure. This incision is also sufficient for the majority of cases of ruptured supraspinatus tendon, but in exceptional cases it may be carried over the shoulder to become the "sabre-cut incision."

FIGURES 2, 3, AND 4

The "Sabre-cut Incision" to Be Used in Exceptionally Bad Cases of Ruptured Supraspinatus Tendon or in Cases of Irreducible Fracture-dislocation



"Sabre-cut" seemed an appropriate name for this incision, for it might well be made by the downward cut of a sabre on top of the shoulder. An incision is made through the acromioclavicular joint and continued with a saw through the base of the acromion. The anterior point of the incision would be continuous with a previous routine bursal exploratory incision. When the acromion has been sawed through, an epulet of tissue, consisting of the deltoid muscle and the acromion process from which it arises, is formed to be pulled outward and downward.

This step is accomplished with ease, for it is only held by a little areolar tissue and a few fibres of the trapezius attached to the upper margin of the detached portion of the acromion. The upper posterior fibres of the deltoid must be separated a little to gain mobility. In sawing the base of the acromion one must bear in mind the suprascapular nerve which supplies the supra- and infra-spinatus muscles and lies between them, a little below the saw-cut. It is deep enough to be out of the way of the saw but not of gross carelessness.

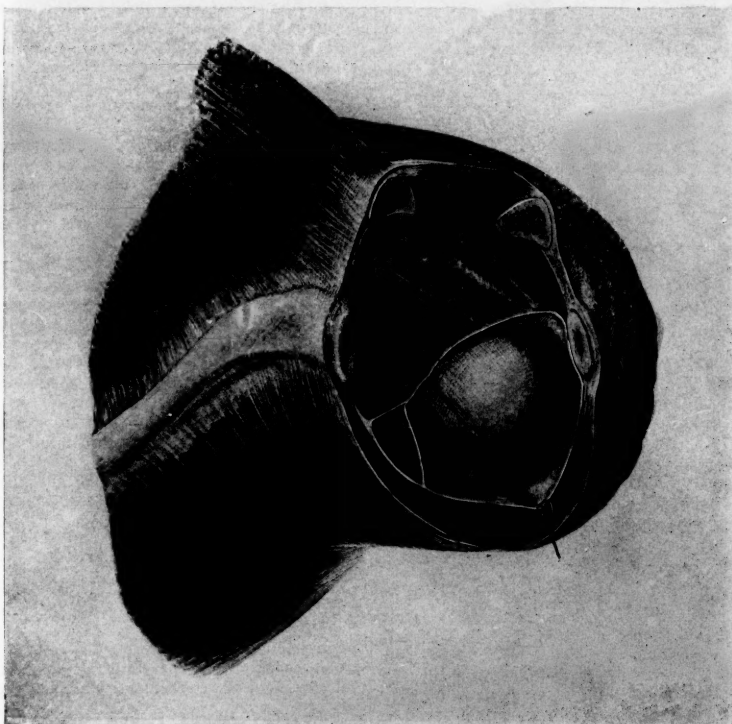
DISCUSSION

DR. DAVID CHEEVER, Boston: I would like to ask Dr. Codman three questions. In the first place, he spoke of the physical examination being negative. I thought he said that in the recent cases there was tenderness immediately below the acromion. Second, I would like to

QUESTION BY MEMBER: I would like to ask if the X-Ray will show any indication of a rupture that isn't severe.

DR. LAMPSON: I should like to ask in what position he puts the arm after operation?

DR. SWETT: I should like to inquire of Dr. Codman whether he thinks of putting the shoul-



The second (Figure 3) view shows the structures exposed when this epulet is pulled downward and outward. Even without dissection one can identify the subscapularis, supraspinatus and infraspinatus as they emerge to join together their tendinous expansions beneath the base of the bursa. To one unfamiliar with this dissection the smooth convex surface of this

base appears to be the articular surface of the humerus. The subacromial and subcoracoid or coraco-humeral bursae are nicely shown. As explained in previous papers, they are often intercommunicating and are always functionally one bursa although frequently, as in this instance, separated by one of the diaphanous nictating folds. Notice the separated portion of the acromion and see how easily it will fit back into place.

know after his exploratory operation whether he expects to repair most instances of rupture through that incision or whether he does the sabre-cut incision. And third, I would like to know something about the mechanism of the causation of the rupture. He speaks of a wrench of the shoulder joint. I always thought it was true rupture by sudden abduction of the arm to save the individual from injury when he falls—that it was a true rupture rather than due to a wrench.

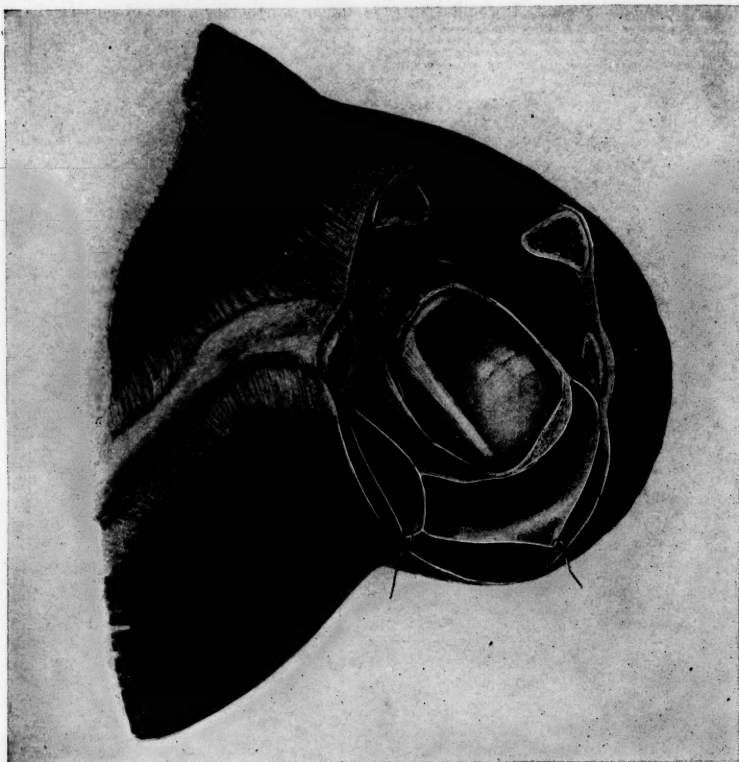
der in extreme abduction in these early, acute cases of partial rupture and what the result of that treatment would be.

DR. E. A. CODMAN, Boston (closing): In regard to the absence of physical signs which Dr. Cheever asked about—I am a little at sea because, as I say, I don't see these cases when first injured. What I see are the results of cases that have been treated by others for six months to two years, so that I don't know about the signs in the acute stage. I do know about them

in the cases I see, and in these I find that other surgeons who know general surgery well have overlooked the physical signs. I think I can detect them. I think I have experience enough so that by putting my finger below the tip of the

but it falls at once. They change their angle of the humerus with the scapula suddenly so as to prevent the arm from falling down and hurting them.

In regard to when the sabre-cut incision is



The third drawing (Figure 4) is identical with the last except that the supraspinatus and capsule have been cut across into the true joint and the ends of the supraspinatus depicted as retracted. The stub of the tendon is still attached to the tuberosity beneath the base of the bursa, while the muscular belly is retracting into the supraspinatus fossa. The glenoid and the articular surface of the humerus are exposed, with the long head of the biceps arising from the superior edge of the glenoid lying across the cartilaginous surface of the head of the humerus.

This is exactly the condition I have found at operation again and again in the living, except that there is seldom so much of a stub of tendon still attached to the tuberosity. Quite frequently it is entirely evulsed from the latter, requiring drilling of the tuberosity to resuture it. I have always found the base

of the bursa to be torn across with the tendon. The point of least resistance appears to be about the subbursal portion of the tendon. In fact the tendon itself is very short, the muscle fibres beginning within a half inch of the attachment.

In the long-standing cases on which I have operated the biceps tendon is found inflamed, swollen and bright pink in color, forming a striking contrast with the white articular surface of the humerus. Sometimes it is apparently absent entirely, having been evulsed and then retracted downward into its sheath.

To close this incision the parts are sutured back into place in the reverse order of these diagrams. It is probably safer to wire the acromion process, although catgut in the soft parts holds it well. I do not advise attempting to close the bursa even in the exploratory operation; a stitch or two in the muscle hold the edges in sufficient apposition and excess fluid may drain into the areolar tissue.

acromion I can elicit a little soft crepitus that I can bank upon. Tenderness is not always present in the old cases but it is probably usually present in the acute cases as well as this peculiar soft crepitus and the jog in motion which is so characteristic. You can raise their arm up,

necessary. Ordinarily I do the simple bursal exploration and ordinarily I can reach in with a tenaculum and grasp the supraspinatus where it has retracted and pull it out and suture it, but occasionally I cannot do this. I cannot get a needle under the acromion so as to get the ten-

don; then I do the sabre-cut. But I regard that incision as chiefly important in old dislocations and fractures, in which cases it is a most satisfactory procedure.

Now as to the mechanism of rupture. I had two characteristic cases in women who threw heavy wet blankets over clothes lines and ruptured their tendons. In a similar way the patella is occasionally broken from muscular violence, i. e. sudden contracture of the quadriceps. I think Dr. Cheever is right that most of these ruptures of the supraspinatus occur from the effort to retain balance as in a man falling, and suddenly throwing up his arm. Sometimes they are co-incident with dislocations and if these are reduced and the rupture not sutured, you get the same bad results.

Is the X-Ray any indication at all? I suggested at the end of my paper that you might inject air or an opaque fluid into the bursa for diagnosis. I have injected air, but it showed so vaguely I couldn't use it in a lantern slide. I believe injecting air just under the acromion is a harmless procedure; perhaps injecting an opaque fluid would be still better.

Now the position after operation—I have treated these cases in various ways. It is hard to keep them comfortable in any way in abduction, but to tie the arm to the head of the bed so that the patient can move it about a little and to pad the arm up with pillows is a good way.

To put a long strap from the head of the bed to the foot and tie the arm to that is also a good way. In some of these old laboring men the discomfort with an arm up that way is too much, so I put them on a splint in a semi-abducted position and let them get up.

Now concerning the partial ruptures where the tendons were only slightly torn off, I believe as Dr. Sweet suggested, many would be improved and their convalescence shortened by rest in bed with abduction. I believe if this were done as a routine in shoulder injuries it would save much trouble and would tend to bring the parts together without being under tension. I believe it would be still better to do the incision into the bursa and put a little stitch into the tendon. You must bear in mind that the bursa lies partly over the tuberosity and partly over the tendon and normally there is a quarter of an inch in cross section between the bursa and the joint. This tissue is composed of the tendon, the capsule, the lower synovial surface of the bursa and the synovial surface of the joint beneath. The rupture when severe extends through all these, putting the joint in direct communication with the bursa. The rupture when severe extends through all these, for the fragment carries the bursa with it. Probably in some cases the bursa remains intact. It certainly does sometimes when the tuberosity is actually torn off by the tendon.

ORIGINAL ARTICLES

HYPERSENSITIVENESS*

BY HANS ZINSSER, M.D.†

I

It is not surprising that there has been much confusion in the clinical classification of the various forms of hypersensitiveness, since even laboratory workers who are occupied largely with the elucidation of these reactions have differed widely in their views concerning them. The original classification of the hypersensitive states suggested by Doerr,¹ as well as the somewhat different but equally useful one of Coca,² both had the logical purpose of drawing a distinction between those manifestations in which, as in protein anaphylaxis, an antigen-antibody mechanism was demonstrable, and the larger group in which no evidences of such a mechanism can be detected. This distinction, like most classifications, served and still serves a useful purpose in furnishing a scaffolding for reasoning. But we have felt from the beginning that they should be regarded as purely tentative guides for in-

quiry, since similarities between the various conditions were striking, and our knowledge of many of them was so vague that failure to elucidate the mechanism in such cases could be attributed as much to experimental limitations as to fundamental differences. In protest against the rigid separation insisted upon by Coca of what he calls the "allergies" from relation to protein anaphylaxis, we wrote in 1923 that "while we were faced with a large variety of hypersensitive states, and while classifications such as those of Doerr and Coca possess considerable preliminary value, it would be a pity if the erection of such fences of division at the present time should influence investigators to overlook fundamental similarity." The purpose of our present paper—since it is written for practitioners who see these conditions in the clinic—is to reiterate this view, since we believe that correct clinical analysis and progress in therapy depend to a large extent upon the correct appraisal of the degree to which all of these conditions are based upon analogous biological laws. Moreover, it has already become apparent that

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†From the Department of Bacteriology and Immunology, Harvard University Medical School.

many of the observations necessary for an understanding of these conditions cannot be made upon animals. It is a peculiarity of hypersensitiveness that identical inciting agents, by a probably identical mechanism, produce widely divergent effects in different species of animals, and many of the problems underlying the clinical manifestations of human hypersensitiveness can therefore be solved only by observation of the human being and by conservative experiment upon clinical material, so far as this may be safely done. Doerr,³ who has made valuable contributions to this subject, and who was the first to formulate their analysis by an attempt at classification, seems now to believe with us that the similarities between these conditions are of more significance than the divergences. Moreover, work of the last few years has tended gradually to diminish the significance of differences which were formerly assumed to be of fundamental importance.

It appears to us that immunologists have taken too narrow a view of the biological significance of altered reaction capacity, and have laid too much stress upon the incidental fact of whether or not specific reaction products, of which we speak as "antibodies," appear freely in the circulation as a consequence of altered cell capacity.

When the phenomena of specific hypersensitiveness were first subjected to experimental analysis, attention was largely concentrated upon sensitization with the coagulable proteins. It was found that the mechanism which was revealed as the responsible one in these reactions did not hold good in all its details for hypersensitiveness in general; and some observers went so far as to express the belief that other forms sickness, hay fever, food and drug idiosyncrasies, "allergies" of Coca, in which he includes serum sickness, hay fever, food and drug idiosyncrasies, horse asthma, etc.) were based upon a mechanism in which antigen-antibody reactions were not involved. Because such a mechanism had not been demonstrated in many of these conditions, Coca—particularly—denied its existence and based a separate group classification of these manifestations upon what he believed was a common association by the establishment of heredity as "the sole determining factor".² In another place he characterizes the fact of heredity transmission as "a positive basis of association which will not only completely justify the association on the ground of common etiology, but will confirm the separation of the phenomena of allergy from those of anaphylaxis." To our view, this extreme attitude, which neglects many similarities in favor of a few differences, is illogical unless these differences can be shown to be fundamental. We do not believe that they are, and it seems to us that such rigid separation is likely to impede understanding of the significance of many clinical manifestations. We will endeavor in re-examining these phenomena with you to indicate why we believe that the basic similarities

ties between the various forms of hypersensitiveness are sufficient to indicate probable dependence upon common fundamental, biological laws.

We may state that hypersensitiveness exists whenever an individual reacts specifically to contact with a given substance with symptoms that fail to develop upon similar contact in a normal individual of the same species. If the substance involved is non-toxic, the normal individual will show no reaction whatever to contacts which may give rise to the most extensive and serious results in the hypersensitive. If the agent is a primarily toxic one, the hypersensitive individual will react to sub-toxic amounts, and the manner in which he reacts may have no symptomatic relationship to the usual toxic effects of the responsible agent.

There is a large range of substances to which animals and human beings may be hypersensitive, a range extending from the proteins down to such chemically definable materials as quinine, arsenic, bromides, iodoform, etc. The symptom complex, however, which is elicited in the sensitive subject by any of these substances depends upon the state of hypersensitiveness of the individual, and upon the location in which the contact between his tissues and the responsible material takes place, and not upon the chemical or physical nature of this material. Thus the clinical pictures of horse asthma, hay fever, etc., are fundamentally alike, though the substances eliciting them are quite different; and the skin eruptions which may follow the ingestion of fish, strawberries, milk or other materials are essentially alike, given an individual specifically hypersensitive to any one of them. It is, in other words, the peculiar susceptible condition of the subject that determines the physiological reaction when specific contact occurs.

In animals the nature of the reactions depends upon species, for reasons that we do not clearly understand. In guinea pigs the emphasis is upon the smooth muscles of the bronchioles; in rabbits, as Coca has shown, upon the smooth muscles of the pulmonary arterial system; and in dogs the liver is particularly involved. In each case, therefore, there is a characteristic clinical focus for the species.

In man the manifestations of hypersensitiveness are manifold, but fall into groups characterized by the localization of disturbances. Roughly, they may be divided into:

- (1) Serum sickness following the systematic administration of foreign proteins.

- (2) Respiratory conditions, in which may be included rhinitis, bronchitis and asthma, which—like hay fever—are particularly associated with materials that reach the body by inhalation.

- (3) Intestinal idiosyncrasies which follow the ingestion of the responsible material and include not only direct disturbances of the digestive organs, but are usually complicated by skin manifestations and sometimes by respiratory attacks.

- (4) A group of skin manifestations which

may occur either alone or associated with other symptoms.

The bacterial allergies we must treat of separately, since they form a difficult chapter in the story of hypersensitiveness which requires for its elucidation a detailed consideration of immunological problems not easily discussed without unduly prolonging our paper.

A number of workers, some of them for convenience of discussion, others because of a belief—wholly erroneous in our opinion—that there are fundamental differences of mechanism, have divided the phenomena of hypersensitiveness into the two groups—true anaphylaxis on the one hand, and the idiosyncrasies or allergies on the other. With differences of nomenclature we will not occupy ourselves, since they are unimportant and turn chiefly upon the proper use of the term allergy.*

Protein anaphylaxis is the most thoroughly studied of the group. The coagulable proteins are true antigens in the sense that upon injection into the animal body they induce specific cellular activity in the course of which there appear in the blood stream substances which specifically react with the injected material. After one, or several, injections of a foreign protein these specific antibodies can be demonstrated *in vitro* by mixing the serum with dilutions of the injected material and observing a variety of effects, among which the formation of a specific precipitate is the most obvious. In view of the specificity of the anaphylactic phenomena, and the entirely analogous specificity of the antibodies, it is a legitimate deduction that there is some connection between the two. It is at least reasonable to infer that the first injection of the protein may have induced specific antibodies which, upon second administration, react with the injected substance within the body in a manner analogous to that in which we know they will react in the test tube. Again, the identity of anaphylactic symptoms in a given animal species—say the guinea pig—whether sensitized and reinjected with egg albumen, horse serum, or vegetable protein, indicates that the characteristics of the seizure are determined in no way by the nature of the sensitizing substance, but are the direct result of the mechanism set in motion within the body by the union between the antigen and its antibody.

*The terminology has been somewhat confused by the fact that the word "allergy" as first used by von Pirquet was meant to apply to all forms of altered reaction capacity of cells and was used in this comprehensive manner by Doerr in his first classifications. Coca, who suggested an alternative classification, applied the term "allergy" to all forms of hypersensitiveness not falling under the strict definition of anaphylaxis—that is, conditions in which an antigen-antibody mechanism had either not been demonstrated or was regarded by him as not playing a role. In this paper we are taking a position in many points considerably removed from that taken by Coca, but we are using the terms "allergy" and "idiosyncrasy" in his sense, since this is the nomenclature which has more or less prevailed in America. Incidentally, we wish to state that while our theoretical considerations differ materially from those of Coca, we believe that the facts both of his own observation and those cited by him in many places from the literature are entirely correct. It is only in their interpretation and estimate of theoretical significance that we differ from him.

The fact that the sensitization can be transferred to a normal animal by injection of the blood serum of a sensitive animal at certain stages and that the quantitative capacity of blood to thus convey passive sensitization is roughly proportionate to its content of specifically precipitating antibodies confirms this belief that the antibodies are responsibly related to the effects.

The interval which is necessary between the injection of an antibody containing serum and the time at which sensitiveness is established in the animal thus injected, coupled with the negative observation that it is rarely possible to produce anaphylactic shock by injecting the reacting substances simultaneously, shows that the union of antigen and antibody within the blood stream is relatively unimportant and that shock depends, at least largely, upon the union of antigen and antibody within or upon the surface of tissue cells. This assumption has been further strengthened by experiments upon isolated organs by methods such as that of Dale which was so astutely utilized for this purpose by Richard Weil.

Further than this in the analysis of anaphylaxis we cannot go; for what happens when this reaction takes place, whether there is a sudden injury to the cytoplasm of the cells in which the reaction takes place, whether mere cell surface reaction throws them suddenly into a state of altered function, or whether, as Dale and—lately—Manwaring suggest, a poison is suddenly liberated, has not been proved. What is likely is that the condition here, just as we shall see in the idiosyncrasies themselves, is a reaction which to a large extent involves the capillaries, causing permeability, local oedema and abnormal activity of some of the cell groups, such as contraction of smooth muscles.

Conceiving the mechanism of anaphylaxis to be such as we have described it, a number of apparently mysterious circumstances become comprehensible. We can understand on this basis, for instance, why an anaphylactic individual will often, after a severe reaction, become temporarily non-sensitive or de-sensitized, since for the time being the capacity of his sensitive cells to react with the antigen has become saturated.

And we would be led to conclude, even without experimental proof to support it, that such a de-sensitized individual must, after a sufficient length of time, return to the sensitive condition, since the de-sensitizing dose has now become merely a further stimulus to the production of antibodies.

Most important of all, though we do not adhere to the categorical statement that intravascular reactions never elicit symptoms, we must bear in mind that it is the cellular antibody elements and not the circulating ones upon which reaction ordinarily depends. We cannot, there-

fore, in all cases expect positive transfer to be successful.

II

Having thus outlined very briefly the basic mechanism of true protein anaphylaxis, let us examine to what extent the allergies or idiosyncrasies as a whole, including all the clinical varieties, correspond to true anaphylaxis.

Most striking of all is the specificity of the phenomena which is true of idiosyncrasies whether respiratory, intestinal or otherwise localized.

Again, the similarity in the nature of reactions within certain clinical groups is independent of the chemical or physical nature of the responsible agent, and is obviously dependent upon the condition of hypersensitiveness of the cells by a mechanism which, if not identical with antibodies, forces the assumption of something fundamentally similar.

Again, as in anaphylaxis, contact of the body by a proper channel with the inciting agent is followed by symptoms with extraordinary speed and with a violence out of proportion to the amounts of the agent involved.

As in anaphylaxis, de-sensitization can be accomplished by a careful and systematic administration of the responsible agent, a matter that has been proved for many idiosyncrasies if not for drug sensitiveness and a few others. This again forces the analogy with antibody-like functions.

And if the clinical desensitization of man is less effective and less easily accomplished than in animals, this is easily understood if we consider that de-sensitization is a quantitative matter and the complete effect cannot be accomplished by a single injection or in a short time, even in an animal, unless we venture an approach to the danger line far closer than we are justified in risking in man.

Such de-sensitization, again, is temporary in the idiosyncrasies as in anaphylaxis, there being eventual return to sensitiveness if the subject is left alone, and as in anaphylaxis, sensitiveness once established may last through life, with perhaps a gradual fading out if new contacts are avoided over a period of many years. In most idiosyncrasies, furthermore, whatever their localization, there is usually evidence of general sensitization, in that both asthmatic and intestinal patients may have skin rashes and general systemic disturbances and in that diagnostic cutaneous tests are equally applicable to a large percentage of all of the cases.

Of the greatest importance, finally, in indicating fundamental similarities among all these conditions is the fact that in all of them, whatever the localization or the differences of species type, the symptomatology appears to be secondary to primary action upon capillaries, with oedema, increased permeability and direct peripheral action upon smooth muscle.

And in considering this apparent capillary mechanism in connection with the fact that, in true anaphylaxis at least, the reaction is instigated by a union of the antigen with sessile antibodies, it is at least interesting to note that several observers, notably Gay and Clark,⁴ have successfully inhibited antibody formation by saturation of the animals, during immunization, with trypan blue—a dye which selects the reticulo-endothelial system. If this tissue, therefore, is the chief seat of antibody formation, it is to be expected that injection of the inciting agent should exert the first effects of its *in vivo* reaction upon the function of these cells.

There is thus a fundamental similarity in basic principles, all of them pointing to a general specific readjustment of the body cells to reaction with a foreign substance. This alone should prevent us from drawing such sharp lines of separation as those which Coca has made and which Cooke⁵ so definitely exposes in stating that the idiosyncrasies (allergy) express the natural hypersensitiveness "of the individual not produced by immunological processes."

III

In justice to the opinions of these workers, however, who have made valuable contributions to the study of these problems, let us examine in detail the differences between true anaphylaxis and the idiosyncrasies which have led to such opinions of a cataclysmic, separate creation of two fundamentally different mechanisms for similar diseases.

One of the most important points upon which a differentiation of the idiosyncrasies is based by workers who are inclined to take this point of view is the fact that although many of the substances which are responsible for idiosyncratic seizures are true antigens, there are others that induce conditions belonging in the same clinical group but which are decidedly non-antigenic in the ordinary sense in which this term is used. This cannot be denied, since it is clear that no antibodies can be induced, however energetic the immunization, by such substances as the various drugs and metals, by tuberculin, Mallein and a good many other inciters of allergy. Moreover, the recent investigations of Grove and Coca⁶ have shown satisfactorily that the complete removal of all nitrogenous substances from pollen and from certain varieties of house dust by tryptic digestion caused no appreciable lessening of the activities of these extracts in sensitive individuals. It is indeed well established that many materials which will not produce circulating antibodies on systemic injection may incite various forms of hypersensitiveness. On the other hand, in reasoning from this we must bear in mind that the clinical conditions incited by such non-antigenic materials are in no important essential different from the conditions incited by other perfectly valid

antigens, and that we have learned a good deal about the antigen function of substances in the last few years which indicates that a substance may still react specifically with an antibody without having the capacity of inducing antibody formation. Thus, for instance, our own studies^{7, 8, 10} with the so-called bacterial residue antigens, as well as those of Avery and Heidelberger¹¹ have demonstrated that Landsteiner's supposition of partial antigens or "haptenes" was a wise prediction, since these substances, simple carbohydrate fractions of the total bacterial antigen, will still react with antibodies without themselves being capable of inducing antibody formation. More pertinent still are the studies of Forssmann¹² and the subsequent investigations of the Forssmann antigen by Doerr and Hallauer¹³, Landsteiner¹⁴ and others indicate that many pure lipoids may be converted into truly antigenic material by simple contact with protein.

We have considered all this in a number of preceding papers⁸, the burden of which in this regard was the thought that the property of inducing the formation of circulating antibodies was probably a function of both the chemical constitution and of the molecular size (diffusibility) of the injected substance; that all circulating antibody-forming materials were non-diffusible and that in the case of the diffusible ones the entire reaction might well be considered as occurring intracellularly without the necessity of antibody formation. While there is of course much that is purely speculative in such reasoning, it is at least logical when we find that practically the only differences between the hypersusceptibility to a protein and that to a diffusible substance lies in those phenomena which depend upon the appearance of circulating antibodies. In our own work on the tuberculin reaction, moreover, we have shown repeatedly that while sensitiveness can be induced in guinea pigs in a specific manner with dead tubercle bacilli^{15, 17}, this sensitiveness has nothing whatever to do with those antibodies which react by precipitation or complement fixation with the ordinary tubercle bacillus antigen, and similar reactions have been demonstrated by us for pneumococcus sensitization. It is thus perfectly clear that while we are dealing with a stimulation of a specific cellular change, the reactions which are stimulated by the cells do not take the form of circulating antibodies. And there are other bacterial constituents, such as the nucleoproteins, which will sensitize and will also produce antibody formation, but only after the most energetic and copious treatment. Thus it seems to us entirely artificial to expect that all materials which are antigenic in the sense that they can arouse specific reactions should participate in that single quality of the coagulable proteins and the unfractiionated bacterial substances of inducing a type of reaction which ex-

presses itself in free discharge of the cellular products into the circulating blood.

Closely associated with this problem of the non-antigenic nature of some of the allergic agents is the question of passive transfer. In typical anaphylaxis, as we have seen, it is a relatively simple matter to convey sensitization from one animal to another with the blood serum, that is, the antibodies, of a sensitized individual. It is the subsequent association between the injected antibodies and the cells, however, which gives rise to the sensitiveness of the subject, and if we understand that it is only the antibodies so associated which play a role in the clinical reactions, we can understand that it will be impossible to carry out such passive transfer from individuals in whom the antibodies are either absent or quantitatively of low concentration in the circulation.

It is true that in most cases of human idiosyncrasy, it has not been possible to transfer passively in the ordinary manner in which this can be done from rabbit to guinea pig and from guinea pig to guinea pig. However, there are a number of exceptions to this rule which seem to break through the wall which separates anaphylaxis from idiosyncrasy on this basis alone. There is the well-known case of Ramirez¹⁸, who transferred horse asthma from one individual to another by blood transfusion. There is the case of Schloss¹⁹ who rendered guinea pigs anaphylactic by injections of blood serum from cases of food idiosyncrasy. There is, furthermore, the recent work of Praussnitz and Küster²⁰, ably confirmed by Coca and Grove²¹, who succeeded in transferring local skin sensitiveness from man to man by injecting the serum intracutaneously into a normal subject and following this by injection of the particular antigen into the same site within 24 hours. De Boche²² also confirmed Praussnitz and Küster's work, and it is significant that he did this with a coagulable protein, namely, horse serum. It is true that such experiments as those of Schloss and similar ones by Longcope and Rackemann²³ and others did not induce in guinea pigs the particular type of idiosyncratic seizure which existed in the individual from whom the blood was taken, but, as Doerr has justly pointed out, this cannot be expected, since the clinical peculiarity of the diseased individual depends not only upon the fact that a sensitization exists, but upon the localization of the reactions, which is determined surely by the manner in which the agent enters the body and not improbably by local differences in sensitiveness of various tissue complexes. It must also be admitted that the experiments of Praussnitz and Küster and those of Coca indicate that the substances which are responsible for the local passive transfer of sensitiveness in their experiments are not identical with the ordinary precipitating antibodies, but this is—in our opinion as in that of Doerr²⁴—or secondary

importance, since—after all—these bodies are functionally similar to antibodies, in that they represent a circulating substance which carries with it the specific power of reacting with the inciting agent, and of lending this reacting power to the tissue cells of the injected individual after contact. Thus they differ from the regular antibodies in no manner, as far as their sensitizing functions are concerned, and our own rather extensive experience with bacterial hypersensitiveness has shown us satisfactorily that the antibodies which ordinarily react with bacteria are in no way responsible for the bacterial allergies. It occurs to us that no useful purpose is served by too narrow a conception of which is meant by an antibody, and that it is of far greater biological importance that sensitiveness to any material is accompanied by the specific formation of reacting substances than whether or not these are or are not given up to the blood stream. It is quite plain that the sensitizing substances of Prausnitz and Küster, to which Coea has given the name of "atopic reagines," are different from ordinary antibodies in some of their secondary characteristics, but in biological principle fulfill similar functions. Coea, indeed, whose observations we have always found accurate, though we disagree from his interpretations, states that the Prausnitz and Küster "antibodies" convey sensitiveness passively, are specific and that local specific de-sensitization can be accomplished. If they differ in minor respects from that type of antibody with which we are most familiar, our limited knowledge of antibodies as a whole cannot permit us to segregate these substances into an entirely different class. Functionally, in regard to hypersensitiveness, they strengthen the analogy between protein anaphylaxis and hay fever and horse asthma.

The point of differentiation upon which Cooke and Vanderveer²⁴ particularly base most of their views is the question of inheritance. They believe that anaphylaxis and the idiosyncratic conditions may be sharply differentiated by the statement that true anaphylaxis is acquired, while the idiosyncratic conditions are inherited by a somatic heredity which follows Mendelian laws. The valuable studies of Cooke and Vanderveer, and similar ones by Schloss and others, establish the importance of the hereditary factor, but in appraising these conditions in their bearing upon the differentiation referred to, it is necessary to consider that the specificity of the reactions is rarely inherited, that offspring may be sensitive to substances quite different from those to which the parents were sensitive, and that the sensitive condition usually appears some time after birth, often not until young adult life. These facts render it necessary to conclude that the cellular function which is inherited is not the established sensitiveness, but nothing more or less than the tendency to be

sensitized. Everyone who has worked with animals, especially in the production of anti-sera in horses, has observed that there is a marked difference in the capacity of individual animals to produce antibodies. This is so marked that many horses are discarded after preliminary tests. The same thing is to a less noticeable extent true of the smaller laboratory animals, and in man, with his varied racial constitution and occupational and dietetic habits, it is likely to be more marked than in the animal kingdom. The rational appraisal of the valuable observations of Cooke and Vanderveer and their associates must, we believe, result in the conclusion that there is a definite inheritance of capacity for sensitization, but that the specific form of sensitiveness is acquired in the course of life by contact. This, indeed, is a supposition that is strengthened by our growing experimental knowledge of the possibility of sensitizing both animals and man through the intestinal and respiratory tracts, and that permeability to antigenic substances on the part of the mucous membranes becomes possible under conditions of very slight deviations from the normal.

Likelihood of the validity of such reasoning is strengthened by the accumulating evidence that true idiosyncrasies can be acquired. Doerr cites the fact that many individuals who are in constant contact with nickel salts or wheat dust eventually become specifically hypersensitive. He quotes Dole²⁵ as finding that two or three percent of all workers exposed to quinine dust become, eventually, hypersensitive, and Rackemann²⁶ and others have cited numerous cases in which circumstantial evidence indicates that horse asthma and the specific coryza induced by Orris face powders have been acquired by use. Evidence is accumulating, therefore, that typical idiosyncrasies can be induced by previous contacts, although in the large majority of idiosyncrasies, as a class, the origin of the sensitization must be—from the very nature of the process—obscure.

The fact that clinical idiosyncrasies may be manifest in many different regions of the body offers no particular difficulties as regards the relationship of these conditions to anaphylaxis. On the other hand, it is a difficult matter to reproduce such localization in the animal experiment. Nevertheless, this has been accomplished. As far as the skin is concerned, many observers have succeeded in obtaining sensitizations which are at least more intense in the skin than in the body generally. Attempts to produce regional sensitization in the lungs and intestine of animals have been less successful. Nevertheless, it is at least reasonable to suppose that a sensitiveness which is probably acquired by ingestion, inhalation or skin contact with a responsible agent, usually expresses itself in local symptoms at the point of first and concentrated contact with the same agent absorbed by the same path. In-

deed, it is not even necessary to assume that the cells at the point of entrance are quantitatively more susceptible than the rest of the body—a thing which, in itself, is likely. It is sufficient to recognize that in the presence of a general sensitiveness the cells at the point of entrance are the first to be saturated with the antigen as it enters the body. Interesting examples of this are our own reactions during a period of large scale tuberculin production, when inhalation of spray induced severe, general systemic seizures which were always preceded and accompanied by violent coughing and acute bronchial irritation. That the reactions between agent and cellular anti-substance might actually take place locally to the exclusion of the rest of the body is amply indicated by Mackenzie's²⁷ interesting experiments in which the local reactivity of the skin could be abolished by repeated applications to the same area of the responsible substance, and in which this specific exhaustion of reaction capacity was strictly limited to the area to which the antigen had been applied.

It is our opinion that in that branch of general physiology which is spoken of as immunology, we have been hampered in our reasoning by adhering too closely to analogies furnished by the study of a few antigenic substances such as bacteria, animal cells and coagulable proteins. We are actually dealing with phenomena of much broader physiological interest. It is perfectly logical to assume, as Ehrlich did, that the tissue cells of higher animals are normally prepared only for metabolic processes concerned in the nutritional and excretory functions essential to the maintenance of life. In this respect, the normal functions of mammalian tissue cells have a relatively narrow range, and the substances which reach the cells for nutritional purposes through the blood and lymph come into actual cellular contact only after preliminary digestion; proteins as amino acids,—lipoidal substances as fatty acids and glycerine and soaps; carbohydrates as simple sugars. The basic lesson that immunology teaches us is the fact that it is only substances of this kind which can repeatedly come into contact with the cells of the body without in some way altering the quality or degree of the cellular reactions aroused by them. It is a biological principle which is most widely demonstrable in the study of hypersensitiveness that most materials which are not in this class of nutritive substances give rise either regularly or under certain circumstances not yet thoroughly defined to a specifically altered reaction capacity; and it is this which von Pirquet spoke of as "allergy" without at the time meaning to imply any given mechanism, and without separating different types. The very facts of hypersensitiveness, with the unmistakable similarity as to specificity, symptomatic complex, laws of desensitization, etc., teach us that such specific cellular reactivity may be acquired against materials separated in regard to

chemical structure as widely as the simple iodides and the complex proteins. And we are forced to conclude that in fundamental principles of cell function there is a common principle underlying all of them.

Have we not been too narrow in our definition of antigen? In the case of the toxins, the coagulable proteins, the bacteria, the red cells, etc., we know that the eventual response of the cells includes the discharge into the blood stream of specific substances which we call antibodies, and which—by the facts of passive sensitization—we assume to be functionally identical with the elements which render the cell itself hypersensitive. In the case of bacterial antigens, and the lipoidal partial antigens of Forssmann, and in the studies of Landsteiner, we have learned that many antigenic substances may be disintegrated, and that part which reacts *in vitro* may retain none of its antibody-inducing properties. In conditions such as hay fever, asthma, etc., where the ordinary methods of demonstrating specific circulating anti-substances have failed, Prausnitz and Küster were able to demonstrate the so-called "reagines," which in all functional properties must be considered antibodies, though following slightly different laws. In bacterial hypersensitivity, our own work has shown that specific sensitization to the responsible bacterial material is independent of the ordinary antibacterial antibodies, and must depend upon a specific cellular reaction substance of a different nature. In many forms of idiosyncrasy, moreover, especially those to drugs, no form of circulating reaction body has been demonstrated, but a cellular specific reaction remains. Is it not just to say that the only difference which exists between the various forms of allergy lies in the fact that in the case of some antigens the specific antibodies become free in the blood stream, whereas in the others this cannot be demonstrated, and—examined in this light—the formation of free antibodies is incidental, and not a fundamental difference, depending probably upon the chemical and physical nature of the antigen, and not upon any difference in the basic mechanism?

From the therapeutic point of view, analysis of the mechanism of the idiosyncrasies has had a relatively modest harvest. In a condition which is so difficult, and in many aspects obscure as this, empirical treatment will always find a fertile field, but reasoning from what we know of the mechanism leaves us with only two clear indications for specific treatment. One of these is de-sensitization, both general, and—as Mackenzie has recently suggested—local, a matter which requires care and judgment. The other consists of prolonged protection against contacts with the sensitizing material, which is feasible only in certain drug and occupational idiosyncrasies, hay fever and a few special ones such as those depending upon feather pillows, fur, face powder, etc. Beyond this we have no

weapons except the logical use of those drugs which antagonize the physiological processes which are immediately responsible for the symptoms of allergy, foremost among which are alterations in the reticulo-endothelial system which lead to capillary permeability.

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CACTINA PILLETS

BY CLIFTON B. LEECH, M.D.*

IN 1923 a study was made of the clinical effect of Cactina pillets (Sultan Drug Co.) upon a group of student nurses and other volunteers. It was known to the author that Hatcher and Bailey¹ fifteen years ago had failed to find that cactus grandiflorus possessed any pharmacologic action, and that at least one scientific investigator had found cactina valueless in 1908².

We were moved to conduct the study, because we were frequently asked by practitioners whether we had used cactina; because some practitioners reported to us its value; because inquiry at local pharmacies revealed that it was sometimes prescribed; because writers in homeopathic journals praised cactus and such journals carried advertisements of cactina; and because the mail regularly brought to our desk matter describing the effect of "Cactina Pillets, a preparation of the Mexican *Cereus Grandiflorus*"—"will strengthen the muscular action of the heart"—"indicated in functional irregularities such as palpitation and feebleness or slowness. They yield splendid results in the support of the heart during chronic and febrile diseases"—"A safe and certain support for the feeble heart."

The dose advised in the advertising matter is "one to three pillets, three or four times a day, according to indications." The indications are "irregular pulse, feebleness of the heart's action; of pressure in the chest; violence of the heart's action—; sensation of constriction or

band around the heart or chest; abnormal heart action, such as tachycardia caused by excessive use of tobacco, tea, coffee, or alcoholics, and in feeble heart following pneumonia, typhoid and other severe and prostrating diseases."

The group study was intended to show any obtainable evidence of physiological effect, subjective or objective. Sixteen persons, ten females, six males, composed the group. All were in excellent health although three presented faint systolic murmurs of the heart. Previous to administration of the pillets a series of observations was made to determine the average weight, heart rate, vital capacity and blood pressure of each subject. Electrocardiograms were taken.

Without otherwise changing the daily routine of the subjects, pillets were administered as follows: for one week one pillet T.I.D.; second week three pillets T.I.D.; third week eight pillets T.I.D.; fourth week sixteen pillets T.I.D. At the end of each week the individuals were examined, questioned in regard to any subjective symptoms, and electrocardiograms were taken. A control group of four was similarly examined each week.

The findings were entirely negative. The heart rate was unaffected. Very slight individual variations in the blood pressure and vital capacity were paralleled in the control group. No effect was evident in the electrocardiograms. There were no subjective findings.

Four persons were given 100 pillets each T.I.D. for one week with the following result.

CHANGES						
	Weight	Heart rate per min.	Vital capacity	Systolic pressure	Diastolic pressure	Electrocardiogram
(1)	+ ½ lb.	-2 beats	0 liter	+4 m.m.	-4 m.m.	No change
(2)	0	-2 "	+1 "	-3 "	0 "	" "
(3)	+ ½ "	-4 "	0 "	-6 "	-12 "	" "
(4)	-7 lbs.	+2 "	-1 "	+2 "	+8 "	" "
Average	-1¼ "	-1½ "	0 "	-¾ "	-2 "	" "

*From the Cardiac Department of the Union Hospital in Fall River.

None of the subjects knew what the pellets were. No one of them reported symptomatic effect upon himself. Several thought the pellets tasted like tea. The writer swallowed 300 pellets at one occasion without experiencing subjective or objective effect.

Despite the lack of evidence of physiological effect upon the healthy it was decided to test the pellets in patients presenting the signs and symptoms for which cactina was recommended. This has been done during the past three years.

Ten patients with extrasystoles were thought to be proper subjects for therapeutic tests as they presented "irregular pulse—palpitation—functional irregularities." None of these patients presented evidence of advanced heart disease and the sole complaint was the annoyance caused by the extrabeats. These patients were given one pellet each day for one week. No improvement occurring, the dose was increased to 2 pellets each day for another week. Then the dose was increased to 3 pellets each day for two weeks. Still there was no symptomatic improvement and no diminution of the extrasystoles. In two of these patients the extrabeats did disappear later upon the cessation of excessive smoking. They reappeared again upon resumption of overindulgence in spite of rations of cactina.

Four patients with constrictive pain in the precordium, presenting no signs of cardiovascular disease, the pain not relieved by nitroglycerin, were given cactina without effect. These patients were cured later by salicylates.

Ten patients with paroxysmal, constrictive,

substernal pain, relieved by nitroglycerin, were given cactina without lessening the number of attacks. Improvement occurred under the usual therapeutics.

Five patients with effort syndrome, presenting sinus arrhythmia and tachycardia, were not influenced by cactina.

Four patients with neuro-circulatory disturbances, fainting and faintness derived no demonstrable benefit from cactina.

Ten patients with myocardial weakness were expected to present definite improvement as the cactina "will strengthen the muscular action of the heart—is indicated in feebleness—is a safe and certain support for the feeble heart." The result was again disappointing. There was no improvement in the heart action or heart rate. Two of this group offered low systolic blood pressures but these were not changed. The slow hearts became no faster and the rapid hearts became no slower.

Thinking that perhaps the cactina had not been used in grave enough cases it was given to two patients with auricular fibrillation and marked congestive failure. No benefit appeared in these so it was given to two cases of regular rhythm with congestive failure, with the same result.

We conclude that cactina pellets are no more than a placebo.

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NEW ENGLAND PEDIATRIC SOCIETY

NOTES ON INFANT FEEDING*

BY KENNETH D. BLACKFAN, M.D. AND JOSEPH JOHNSTON, M.D.

DR. BLACKFAN: Within the past five years a wave of enthusiasm for the use of acidified milk in feeding both sick and well infants has spread over the country. One reason for the almost universal adoption of this so-called "new" method of nourishing infants is the fact that its reappearance at this time was heralded by the announcement that its success was based upon a sound scientific foundation. Marriott, in popularizing lactic acid milk, claimed that its beneficial effect depended on altering the buffer value of the food given so that the pH of the gastric contents reached the theoretical optimum necessary for complete digestion of cow's milk.

That the scientific basis claim impressed the medical profession is demonstrated by the fact that in the Infants' Hospital, during the year 1925, practically every undernourished infant, including the premature babies, received lactic

acid milk. That they thrived is not to be denied; that the excellent results were alone attributable to the acidity, however, seemed questionable when Doctor Johnston pointed out that in the lactic acid milk mixtures the following features might be equally as important as the acidity:

1. Undiluted cow's milk was used.
2. The mixture was concentrated; namely, 1 c.c. equalled 1 calorie.
3. The proportion between the calories furnished by the milk and those furnished by added CH were in just the relation to each other that Powers had recently pointed out as having been the most uniformly successful combination in cow's milk feeding throughout the world; that is, 60% of the calories were in milk and 40% of the calories in added CH.

Doctor Johnston suggested that, if the above-mentioned variables were kept constant and only

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the acidity varied, equally good results might be obtained with unacidified milk as with acidified milk. Marriott and Davidson mention such a study of ten babies, and report results which, in terms of average daily gain, were almost two to one in favor of the acidified milk. Our study was essentially a repetition of their work in an attempt to confirm it and especially in an attempt to question the usual assumption that undiluted cow's milk balanced with added CH is not tolerated by infants.

During the past year, with these points in mind, we have endeavored to see if we could demonstrate any superiority, as determined by gain in weight, character of the stools, and general condition of the infant, of acidified milk over unacidified milk. The pH of the gastric contents was determined in a number of instances. As is apparent, the problem suggested was less one of scientific study of acidity than a simple application of logic. It should be emphasized that the infants selected were of the hospital type, suffering with severe nutritional disturbances. The attempt was made to keep constant the three variables mentioned and to control the observations as carefully as though we were conducting experiments in the laboratory. At first only selected patients were chosen; later, the feeding was extended to an unselected group and to prematures. Doctor Johnston will present to you the results of these observations.

DR. JOHNSTON: One of the largest groups of babies which seemed to be ideally suited for the feeding of lactic acid milk with added carbohydrate has been the group brought to the hospital with severe grades of malnutrition. We make no claim to a knowledge of the best method of feeding this type of baby, but it is fairly generally accepted that to gain he must receive a number of calories in excess of those calculated on his actual weight. This number tends to approach those which he would receive if they were calculated on his expected weight. Now if the capacity of the stomach increased proportionately to the increased need for more calories, the infant's requirement might be met by increasing the quantity of food, but the capacity of the stomach tends to remain commensurate with his actual weight. This discrepancy between the increased caloric need and gastric capacity constitutes one of the indications for concentrated feeding. We feel that the success of lactic acid milk is attributable to this element, concentration; but as has been pointed out, if the mixture be concentrated, it will be unnecessary and it may be undesirable to acidify the mixture.

Two other points urged in favor of acidified milk were that higher calories might be fed and a higher carbohydrate added "with safety" than with milk not de-buffered. In the group of infants observed, it was sometimes necessary to feed as high as 90 calories per pound, and the

added carbohydrate varied from 30% to 50% of the total calories. To neither of these points could untoward results be attributed.

From some work on acidity several years ago it had seemed to us that there was an apparent correlation between the pH and the emptying time and that some influence on vomiting might result from the use of acidified milks. We have since felt that perhaps we confused cause and effect. At any rate, clinically, in three of the infants in the group which had been diagnosed as having pylorospasm, we were unable to show that the pH of the mixture fed affected vomiting. The other principles followed in the treatment of these infants—concentration, thickening, etc.,—seemed to exert what effect they did independently of the reaction of the mixture.

An analysis carried out some time ago has some interest in this connection. A comparison was made between the results obtained by feeding conventional dilutions of whole milk with added CH to a group of babies admitted because of failure to gain, in whom disease and organic lesions had been ruled out, and by feeding to a similar group a few years later acidified milk mixtures with little or no added water. The results were altogether in favor of the acidified milk mixtures and the figures representing average daily gain were approximately the same as those obtained by Marriott and Davidson in the group of ten babies on whom they made comparative studies. However, the conventional whole milk dilution differs radically from the acidified milk mixtures in concentration, the number of calories to the c.c. being greatly less. An analysis of approximately 1000 figures representing average daily gain, comparable because the variables were kept constant, gave figures for average daily gain on acidified and unacidified milks which were almost the same—0.61 oz. per day on sweet milk mixtures and 0.59 oz. per day on acidified milk mixtures.

We should like to emphasize again that we are not attempting to put forth any new type of feeding. Schick and von Pirquet and Findlay have all used with apparent success concentrated mixtures in which they felt that the lack of a diluent did not demand acidification. We are merely making the clinical point that we have been unable to see in acid milks any advantage over milks which have not been acidified in a fairly extensive observation on a large group of hospital types of babies, including prematures, and that acidified and unacidified mixtures may be interchanged without perceptible difference being noted, at least at short range, in equally high caloric amounts and with as much added CH. We feel that, if the routine use of lactic acid milk accomplished anything in improving the treatment of athreptic babies, it did away with the popular fear of concentration.

There may be definite indications for the use of acidified milk mixtures, but it is also true that there may be contraindications for their use. Faber has recently pointed out that prolonged use of acidified milk mixtures is accompanied by undesirable changes in the mineral body metabolism. He emphasizes his belief that

acidified milk should be used as a *therapeutic food* and not be adopted as a routine in the feeding of normal babies.

(Demonstration of chart showing weight curve on two types of mixtures plotted over food intake, calories per unit of weight being kept constant.)

VARIATIONS IN THE DIASTASE OF THE BLOOD OF INFANTS

BY GEORGE GUEST, M.D.

A STUDY of the diastase activity of the blood of infants was undertaken with the hypothesis that in infants suffering with various types of nutritional disturbances there should be found significant changes in the activity of those enzymes which are presumably responsible for the utilization of food substances.

Method: A modification of the method recommended by Meyers and Killian for measuring blood diastase activity was used, the method requiring 4 c.c. of whole oxalated blood. Measurement is made of the glucose liberated by the action of 2 c.c. of blood upon 1 c.c. of 1% starch solution; from this measurement an index is obtained which represents the mg. of glucose which would be liberated in a proportionate mixture of 100 c.c. of blood and 50 c.c. of 1% starch solution, under similar conditions.

In eight new-born infants the starch-splitting power of blood taken from the cord was almost nil. For healthy infants of two months the average index was 20, and for those approaching two years, 35. Repeated examinations of

the blood of the same infants at intervals of several days gave remarkably constant values so long as the infants were doing well. In certain pathological conditions marked variations from average normal values were observed.

In so-called "intestinal intoxication," the average index was 10 in fifty cases, and the lowering of the starch-splitting power of the blood was proportional to the severity of the condition described clinically as "intoxication and dehydration." Diarrhea did not appear to play a role in influencing the changes. The blood diastase activity rose coincidentally with clinical signs of improvement and fell again in certain cases showing an exacerbation of the state of intoxication.

In a miscellaneous group of pathological conditions, secondary anemia, tuberculosis, syphilis, meningitis, rickets, and scurvy, normal values have been obtained.

In acute upper respiratory infections and pneumonia extremely high values were obtained, the highest being 146 and the average 58 in sixteen cases.

OBSTETRICAL INJURY OF THE SPINAL CORD

BY BRONSON CROTHERS, M.D.

CASES of obstetrical injury of the spinal cord are not uncommon. During the last six years we have seen approximately thirty. Thus the condition outranks in numbers many of the rarer, but better known syndromes. The chief point to be stressed is that these injuries can be profitably studied by laboratory methods.

The four cases shown represent different physiological pictures. The physiological residue rather than the pathological lesion is the important factor. Since prevention is in the hands of the obstetrician and since the lesion is practically a fixed one, the problem comes down to the utilization of everything which is still intact.

Complete thoracic transections with an intact lumbar cord divide a baby into three parts. Above the lesion a normal child, in the center a flaccid trunk, and below the lesion a reflex mech-

anism. Active reflexes of this type are useless, in fact they complicate rather than simplify the problem since they are completely out of control. On the other hand, it is possible to fit apparatus since trophic ulcers do not occur.

A rather more discouraging type is found in cases where the lumbar enlargement is destroyed. Trophic disturbances prevent adequate treatment by apparatus. Milder grades of injury give rise to scoliosis, spasticity, ataxia and so on.

The cases shown illustrate that these children do not die early. Some of them have lived for nine years with total transection. Furthermore, persistent and resourceful work by the orthopedists has enabled most of our older cases to get about. The bladders do not necessarily become infected. Since these children are intelligent and live for indefinite periods, they are worth taking care of.

BOSTON MEDICAL HISTORY CLUB

THE EARLY PHRENOLOGICAL SOCIETIES AND THEIR JOURNALS*

BY JOHN F. FULTON

THE intellectual outburst created by the doctrines of Gall and Spurzheim may appropriately be compared with that which occurred after the discoveries of Galvani (1791) and Volta (1799). At that time men in every corner of the globe attempted to see how the mangled limbs of frogs could be brought once more to life by means of electric currents. New visions were entertained of vital forces and powers, and for a short time discussion and speculation knew no bounds (du Bois-Reymond). So it was with phrenology. It attracted immediately to its study many of the credulous, but also some of the best intellects of the age. They formed themselves rapidly into societies for discussion of the new and suggestive hypotheses of the continental authors, and journals were soon established to communicate to the world the results of their deliberations. Some conception of the tremendous and widespread interest aroused in the subject may be gained from a consideration of these early societies and their journals.

Spurzheim first lectured in Great Britain in March, 1814, and it was then that interest in phrenology among English-speaking people may be said to have arisen. Seventeen years later, that is in 1832, there were twenty-nine phrenological societies in Great Britain (Macalister, *Ency. Brit.*, 13th ed.) and at least ten in America. There were certainly six journals in English (and possibly there were more) devoted to the advancement of phrenology. In addition, phrenological societies which issued journals sprang up in Paris, Copenhagen and Heidelberg. It is now exceedingly difficult to trace these early societies, since in many instances no printed record is left of their proceedings. If they could all be traced, the number of groups which met for discussion of this fascinating subject might be multiplied indefinitely.

The Edinburgh Phrenological Society.—Undoubtedly the most influential of these associations was the Edinburgh Phrenological Society. Something in the Scottish character, innate curiosity and piety perhaps, together with tenacity of purpose, led many of this race to become aggressive protagonists of the new philosophy. Spurzheim is said to have remarked in his Edinburgh lectures (*Amer. Phren. Journ.*, vol. 1, p. 41, 1839): "You are slow but you are sure; I must remain sometime with you, and then I will leave the fruits of my labours to ripen in your hands. This is the spot from which as a

centre, the doctrines of phrenology shall spread over Britain." We may now describe briefly the circumstances which led to the founding of the Edinburgh Society.

In June, 1815, Dr. John Gordon published a savage attack upon Gall and Spurzheim in the *Edinburgh Review*. It happened that Spurzheim, having left London, was at that time in Dublin, and he came immediately to Edinburgh to defend himself. He gave public dissections of the brain, and a course of lectures* which were attended by many including George Combe. Combe was greatly impressed by all that was said, and later remarked (*Introduction to American Lectures*) that after three years' study he became an ardent disciple and then a prominent expositor of the phrenological doctrines. Leslie Stephen has slyly remarked, "The conversion was probably quicker" (*Dictionary of National Biography*). At all events, Combe visited Spurzheim in Paris in 1817 and returned a thorough believer. In 1818 he began a series of essays on phrenology in the *Literary and Statistical Magazine*, and in the following year wrote a book entitled *Essays on Phrenology*. On February 22, 1820, George Combe, his brother Andrew, and several others of the confraternity, met together and established the Edinburgh Phrenological Society (*Life of Geo. Combe*, C. Gibbon, 1878). The transactions of the first three years were published in a single volume entitled *Transactions of the Phrenological Society of Edinburgh*, in 1824. In December, 1823, the first volume of the *Phrenological Journal and Miscellany* was issued, a work destined to pass through twenty volumes largely under the editorship of George Combe. The society continued to meet at regular intervals until shortly before Combe's death in 1858. Among the early members were Dr. John Abernethy, Sir George S. MacKenzie, Sir William Ellis, Dr. Robert Maenish, Rev. David Welsh, and Dr. Andrew Combe.

The early controversies of the phrenologists are undoubtedly familiar to you. The scurrilous attacks launched against them in the *Edinburgh Review* and *Blackwood's* were usually more bigoted than reasonable, while the enthusiastic rejoinders of the confraternity were characterized in many instances by a "will-to-be-

*Being a paper read to the Boston Medical History Club, January 28, 1927, at the meeting in honor of Spurzheim at the Warren Museum (Harvard Medical School).

*[Spurzheim] "Prospectus of the anatomical views of Drs. Gall and Spurzheim on the brain and nerves, confronted with the *Edinburgh Review* [No. 48, June, 1815, Art. X] and Dr. Gordon's opinions in his system of human anatomy and surgery, vol. 1, Edinburgh, 1815." 8°. Edinburgh [1815?]. Also: Spurzheim, "Examination of the objections made in Great Britain against the doctrines of Gall and Spurzheim." 8°. Edinburgh, 1817.

lieve," being often shallow in logic but always sincere.

The Boston Phrenological Society.—The Society in Boston was also founded as a direct result of the influence of Spurzheim. Spurzheim had sailed from Havre on June 20, 1832. He arrived in New York on August 4, and on the 20th of August reached Boston, where, on September 17, he began his course of (18) lectures on phrenology. Late in October he was taken ill with a fever to which he succumbed on the 10th of November (1832). Seven days later (the day of Spurzheim's funeral) a group of "Boston gentlemen" met in the printing house of Marsh, Capen and Lyon, being presided over by Dr. Tuckerman, and passed, at Dr. J. D. Fisher's suggestion, the following resolutions (Capen, *Reminiscences of Dr. Spurzheim and George Combe*, 1881, p. 119):

"1. Resolved, That we form ourselves into a society to be called 'The Boston Phrenological Society,' instituted for the purpose of investigating the science of Phrenology and its bearings upon the physical, intellectual, and moral conditions of man.

"2. Resolved, That a committee of five, consisting of Hon. John Pickering, Dr. Jona. Barber, Dr. Saml. G. Howe, Rev. John Pierpont, and Wm. B. Fowle, Esq., be appointed to draft a Constitution and By-Laws for the government of the Society, and that the said committee shall have power to invite others to act with them."

The first officers of the Society, elected December 31, 1832, were the following: Rev. John Pierpont, President; Dr. Jona. Barber, Vice-President; Dr. Samuel G. Howe, Cor. Secretary; Nahum Capen, Recording Secretary; E. P. Clark, Treasurer. *Counsellors*—Dr. J. F. Flagg, Dr. Winslow Lewis, Jr., Dr. Jos. W. McKean, and Wm. B. Fowle. *Curators* (elected in 1834)—Dr. N. B. Shurtleff and Henry T. Tuckerman.*

The activities of the Boston Society have been well described by its first secretary, Nahum Capen, in his *Reminiscences of Spurzheim and Combe* (1881). In addition to regular meetings during the year, they had an annual meeting on December 31 of each year in commemoration of Spurzheim's birthday. The annual addresses were issued in printed form in 1833, 1836, 1837, 1838, and 1839, and they are the only contemporary transactions of the society which were published. The address of Elisha Bartlett, delivered in 1838, is especially noteworthy and will be mentioned below. The society ceased to hold regular meetings in 1842.

Mere recital of the details concerning the

early phrenological societies is of little interest without some knowledge of the personality of the members. What manner of men were these early followers of the Gall and Spurzheim? If we take the Combes in Edinburgh, and Elisha Bartlett and Nathan Allen in this country, as examples, we can form a fair estimate of their character.

Andrew Combe was a deeply-beloved Scottish physician, not exactly of the "old school,"—not a "Weelum MacLure,"—for Combe was a leader in experimental physiology; yet he combined in an admirable way the endearing qualities of the country practitioner with those of the skilled laboratory worker. I have here Combe's edition of Beaumont on the Gastric Juice (shown). Beaumont, you may recall, had published his celebrated observations on Alexis St. Martin in Plattsburg in 1833. Andrew Combe, who had himself worked upon the physiology of the stomach, was probably the first in Britain fully to appreciate the great value and originality of Beaumont's observations, and, without a suspicion of jealousy, brought out in 1838 this Edinburgh edition of Beaumont's classic work, copiously annotated from his own experience. Andrew Combe was the victim of pulmonary tuberculosis, and for twenty years he struggled courageously, though vainly, to overcome it. Many of you I am sure are familiar with the delightful essay on Andrew Combe (*Horae Subsecivae*, 3rd series) by Dr. John Brown, the author of "Rab." Throughout his life Andrew Combe was an enthusiastic supporter of the Phrenological Society and its *Journal*, but always in the scientific spirit. He realized the value of experimental methods and was one of the earliest to give an accurate clinical description of the mental symptoms resulting from a small lesion of the brain ("On the effect of injuries of the brain upon the manifestations of the mind." *Trans. Phren. Soc.*, Edinburgh, 1824).

His brother, George, was a less attractive character. He was more of a missionary and less of a scientist, and had less appreciation for the value of experimental methods and logical arguments. Stephen (l.c.) has said of him "He was essentially a man of one idea. His want of scientific training predisposed him to accept with implicit confidence the crude solution of enormously complex and delicate problems propounded by the phrenologists, and for the rest of his life he propagated the doctrine with the zeal of a religious missionary." Nevertheless, George Combe had unusual literary gifts, and his philosophical and phrenological essays are still delightful to read on account of their beautiful English style.

Combe was largely responsible for fostering the *American Phrenological Journal and Miscellany*, which was founded in 1839 with the

*In the list of members one finds the names of many well-known citizens of the time of Boston: Rev. Henry Ware, Jr., Rev. Dr. Brownson, Hon. John Pickering, Hon. Abbott Lawrence, Hon. J. W. Edmunds, Wm. P. Mason, Nathl. C. Nash, Samuel Downer, Chas. G. Loring, J. H. Walcott, Moses Kimball, George G. Smith, Jonas Chickering, Joseph Tilden, Otis Everett, Jr., James Blake, Hon. James D. Greene, Hon. J. S. Sleeper, J. W. Ingraham, E. L. Frothingham, Wm. A. Alcott, Dr. Daniel Harwood, Wilder S. Thurston, Wm. Hunt, F. Skinner, John Appleton, Dr. Henry G. Clark, John H. Bates, Danl. F. Child, Alvan Fisher, Danl. S. Smalley, Dr. M. S. Ferry, Dr. John Flint, John J. Dixwell, etc.

Edinburgh Journal as its prototype. O. S. Fowler, the original editor, turned over his duties after the first year to Nathan Allen, the medical student from Lowell, Massachusetts, who at that time was distinguishing himself in the University of Pennsylvania Medical School. The *Journal* was first issued in 1839, and in 1840 Allen, then a youth of 26, became its sole editor, and for two and a half years it prospered in his hands. Marriage or a desire to enter into general practice, one cannot be sure which, led him to abandon the *Journal* in 1842, when he returned to Lowell and practiced there for the rest of his life.

The *Journal* after Allen left it was taken over again by the Fowlers, but its literary excellence soon deteriorated. Nevertheless, it continued to exist under various titles until 1911 (*American Phrenological Journal and Miscellany*, 1839-50; *American Phrenological, and Repository of Science, Literature and General Intelligence*, 1851-54; *The American Phrenological Journal and Life Illustrated*, 1861-69 and 1871-84; *The Phrenological Journal and Packard's Monthly*, 1870, and *The Phrenological Journal and Science of Health*, 1884-1911).*

Nathan Allen's personality, though in some respects unique, possessed many characteristics in common with other phrenologists of the time. He was given to broad generalization, seeking to find laws and universal principles to "explain" human activities, often without first obtaining requisite facts, and his writings in consequence have at once both the lure and the weakness of his motives. Much the same criticism might be applied to the writings of George Combe, and even to Spurzheim, but less appropriately to Elisha Bartlett.

This busy practitioner and medical humanist,

*I wish to acknowledge my indebtedness to T. J. Homer, Esq., who kindly placed at my disposal the cards of his *Guide to Serial Publications* now being published by the Boston Public Library.

of whom Osler (see *Alabama Student*, 1908) has written so delightfully, was probably the most distinguished physician in America in the early 19th century. In 1838 he consented, as already mentioned, to give the Annual Address to the Boston Phrenological Society, which was clear, preëminently sane and of rare literary excellence.

"Phrenology," he says, "in so far as it claims to have demonstrated the existence of a multiplicity of cerebral organs, each concerned in the manifestation of a primary and elemental faculty or power of the mind, must rest for support, singly and exclusively, on observation. The truth of this fundamental proposition of the science, we believe, has been so established."

Nowhere has Bartlett exhibited to greater advantage his skill in the art of writing. Such passages as the following will serve as an illustration.

"They are the great men of the earth, who have lived the most, and lived with their highest faculties. They too are the good, the truly noble, the royal priesthood, the crowned and sceptred hierarchy of humanity. And so shall they, one day, come to be regarded; so shall be judged the living, and so too shall yet be judged all the historic names of the past. They must all go through this new ordeal, and fiery, indeed, will it prove to most of them, for by it shall they be utterly consumed. Many of the demi-gods of the earth will be found to be idols of clay only, and they will crumble in pieces. The world knows little of its greatest men."

And again:

"Like these flowers that give out their richest fragrance when crushed by the heel of the traveller, many of the best virtues of the soul are strengthened and unfolded by what are commonly regarded as the adverse circumstances of our lot."

Little wonder that one who could think and write in such terms, and who throughout his life had made his brain do its work, was a man after Osler's heart.

THE SPURZHEIM COLLECTION OF PHRENOLOGICAL CASTS*

BY WILLIAM PEARCE COUES, M.D.

ARISTOTLE wrote of the brain as "the coldest and most bloodless of bodily organs, of the nature of water and earth, whose chief purpose is to temper the excessive heat of the heart, as the cooler regions of the firmament condense the vapors rising from the earth." To modern minds the slow and halting unwinding of the mysteries of cerebral function through the ages, seems almost inconceivable, but second thought and historical retrospect give adequate reason for much that tended to obscure knowledge of cerebral function and its relation to mind. The important work of Gall and Spurzheim in relation to neurology, and especially cerebral phy-

siology, was lost sight of to a large extent, being clouded and obscured by their life-long intensive pursuit of the phrenological myth.

For the illuminating history of the remarkable collection of casts now in the Warren Museum, some of which are displayed for your inspection tonight, we are indebted to Dr. J. C. Warren, who wrote an instructive account of the Spurzheim collection in an article published in the *Annals of Medical History* in 1921. To this account I have turned for many facts concerning the collection.

When the Medical School moved its quarters to Boylston street, in 1880, the collection was left behind and was stored for years in the basement of the Grove Street building. After the

*Read at a meeting of the Boston Medical History Club on January 28, 1927.

Dental School was moved to its new site on Longwood Avenue, the building remained vacant, and was at last torn down, when the new Moseley building was erected.

At this time, the late Dr. Reginald Fitz called Dr. Warren's attention to the danger of the collection of casts being destroyed; the collection was, therefore, transferred to the administration building of the new School. Later the casts were cleaned and transferred to the Warren Museum, where you view them tonight, together with the skull and cranial cast of Spurzheim.

Phrenology, a name coined by Foster, was also known as the science of cranioscopy, craniology, physiognomy, and zoönomy. The present interest in the name of Gall is not on account of this exploded pseudo-science, but on account of the extremely valuable work of Gall in neurology and physiology, in which he was so ably assisted by Spurzheim. As we shall see later, somewhat more in detail, this work was lost sight of and overshadowed at the time, on account of the ideas he championed concerning phrenology. When we think of this word at the present time it certainly does not "give us a thrill," as is said in the language of the day; rather does it connote to our minds certain times—the time of anti-macassars, heavy and ugly furniture, stiff shirts that opened in the back, false cuffs and pantalettes, in short the Victorian era, and in the country of the Lady of Windsor the pseudo-science flourished for a time like the green bay tree, then lapsed, Victorian, but not victorious.

The Hon. Nahum Capen (Spurzheim's Fidas Achates during his visit to Boston, and his faithful friend through his last illness, which occurred here), gives us a most interesting account of Spurzheim, and also of Dr. Gall, in his "Reminiscences." John Gaspar Spurzheim, Capen tells us, was born on December 31st, 1776, at Longwich, a village near Treves. His father was a farmer and was employed on the lands of the Abbey of St. Maximin de Treves. Spurzheim's father was a Lutheran, and the son was educated for the church. He matriculated at the University of Treves in 1791. In 1792, after the French invasion, Spurzheim went to Vienna and took up the study of medicine. It was in 1800 that his acquaintance with Gall began, which was destined to last through life, the two travelling and lecturing together for many years. Spurzheim says, "I was simply a hearer of Dr. Gall till 1804, at which period I was associated with him in his labors." We are told that "the moment that Spurzheim became associated with Gall, the anatomy of the brain assumed a new character." In 1802 Dr. Gall's lectures were stopped in Vienna, by decree of the government. After this the two physicians started on their lecture tours, which took them all over the continent, and to England and Scotland. In England, notwithstanding considerable opposition from members of the medical faculty, the new science obtained great popular vogue

and many phrenological societies and journals were started.

A few words concerning Gall are in order, as to the causes which led to the new theories that he championed, and that provoked so much discussion. François Joseph Gall was born in Tiefenbrunn in the Grand Duchy of Baden, on March 9th, 1758. His father was a well-known merchant, and, like Spurzheim, the younger Gall was intended for the church, but he was opposed to becoming a priest, and studied medicine instead of theology, entering the university in 1781.

Gall tells us that from youth he was interested in physiognomy, and was an accurate observer of the different mental characteristics of his brothers and sisters and his other playmates. When he was older he made careful studies of the so-called "natural talents" and also of the failings of many of his fellow students and other associates. He observed that his school fellows who were gifted with good memories and talent for learning all had prominent eyes. He then postulated the idea that if word memory of unusual character was accompanied by this unusual sign, other intellectual powers might be accompanied by non-varying characteristics in external cranial development. He therefore made exhaustive studies concerning this question, studying alike the normal and the sick, and also animals. His position at the asylum in Vienna was used to extend this study along these lines, and he was allowed to examine the skulls and brains of many who died outside of hospitals. These studies and his theories he outlined to De Retter in his celebrated letter on "The functions of the brain in man and animals."

Perhaps no short account of Gall gives a better idea of what he had accomplished and what physiology and neurology really owe to him than that of Elliot Smith in his essay on "The Evolution of Man." In his chapter on the human brain he speaks as follows. "What, however is impressive is the fact that the vast stream of books and memoirs has brought us so few indications of any serious attempt to probe into the really vital issues regarding the way in which the brain has acquired its highest powers. It is barely a century since the knowledge of the structure and function of the brain has reached the stage that permitted really profitable discussion of its distinctive attributes in the human being. The great revolution in attitude was affected by Gall, whose services in the advancement of science are now almost completely disguised by the notoriety associated with his name as the inventor of what afterwards became known as phrenology. Yet it would not be wholly true to say of Gall what Shakespeare put into the mouth of Mark Antony when he said of Caesar:

'The evil that men do lives after them,
The good is oft interred with their bones.'

The evil part of Gall's teaching has undoubtedly lived after him, but the good attained what Huxley, long ago, called 'the euthanasia of scientific work'; it has been accepted as part of our heritage of knowledge even though the credit due to Gall for a great reform has for most of his successors been 'interred with his bones.' It was he who destroyed the ancient speculations concerning vital spirits dwelling in the ventricles of the brain. He proved that the white substance was fibrous, and introduced the method of exposing fibre tracts by dissection to demonstrate their connections with the nervous system."

Elliot Smith further tells us that Gall called attention to the true significance of the grey matter of the brain, and was the first to give the correct account of the connections of the optic tracts, and that the discussions arising out of his claims for cerebral localization provided the stimulus which was responsible for a profound revolution in cerebral physiology, though Gall was so angered by the attacks of the physiologists as to deny the value of experiment.

"In his own lifetime it was the anatomical facts so easily susceptible of confirmation that were most violently assailed by his critics; hence the speculative and untenable part of the doctrines usually associated with his name escaped criticism, and 'lived after him.' But his really great discoveries became tacitly absorbed into the great body of knowledge, in later decades, when men were hot in pursuit of the fallacies of phrenology. Although it was not more than thirty years after Gall's death that the idea of localization of function in the cerebral cortex began to be seriously entertained, chiefly under the influence of Hughlings Jackson and Broca, much valuable work was accomplished in the first half of the 19th century."

Smith further points out that even before the time of Gall's death, the question of the difference between the brain of man and that of the lower animals had been studied. He tells us that Sir Richard Owen, in 1867, revived the claim that the brain of man was differentiated by the presence of the hippocampus minor, but this old assumption had been really disproved by Serres as far back as 1827, and by Huxley and Flower in 1862, showing that "the hippocampus minor was not distinctive of man, as hitherto had been supposed, for it is present in apes and seals. It is an amazing episode in this subject that Owen should have resuscitated a fallacy which had been so utterly demolished as this claim had been, but the incident was not without its uses, for it stimulated Huxley to revise the anatomy of the occipital end of the cerebral hemisphere and so prepare the way for the particular line of research with the latest result of which this address is in the main concerned. Incidentally, also, it inspired Charles Kingsley to write in 'Water Babies' one of the most ironical parodies of a scientific discussion

in the English language. The controversy excited by Sir Richard Owen's contention that the great distinctive feature of the human brain was the possession of a structure that used to be called the hippocampus minor was not unjustly the remark of Kingsley's seathing satire." The quotation from "Water Babies" follows:—

"The professor has even got up at the British Association and declared that apes had hippocampus majors in their brains just as men have, which was a shocking thing to say, for if it were so, what would become of the faith, hope and charity of immortal millions? You may think that there are other more important differences between you and an ape, such as being able to speak, and make machines, and know right from wrong, and say your prayers, and other little matters of that kind, but this is only child's fancy, my dear. Nothing is to be depended upon but the great hippopotamus test. If you have a hippotamus major in your brain you are no ape, though you had four hands, no feet, and were more apeish than the apes of all aperiens. Always remember that the one true, certain, final and all important difference between you and an ape is that you have a hippotamus major in your brain and it has none. If a hippopotamus was discovered in an ape's brain, why, it would not be one but something else."

To return, in conclusion, to Spurzheim and his brief visit to America. Shortly after his wife's death in Paris, which occurred in 1829, and was the end of an ideally happy marriage, Spurzheim received invitations from Boston and other cities in America to lecture on phrenology, which was causing much interest in the new world. He accepted the invitations and sailed for New York in June 1832. After a short time spent in New Haven and Hartford, he arrived in Boston in August of that year. In reading the different accounts of Spurzheim's brief sojourn in Boston, notwithstanding that his thoughts and theories concerning the so-called "new science" were not always agreed with, one is impressed with the fact of the universal respect which his gentlemanly bearing, modesty and charm of manner commanded. In money matters he was almost unbelievably careless, and insouciant. One day he remarked to Capen, "I believe I have some money in my trunk; please take care of it for me." Capen says, "I found nearly five hundred dollars in gold in the bottom of the trunk, but I could not persuade him to count it." Spurzheim fell ill in October of the same year he arrived in Boston (1832), but kept up for some days his lectures, which were largely attended. The disease was, in all probability, typhoid. He would not see a physician for some time, but finally consented to have Dr. James Jackson attend him. It was, however, too late. Had he given up at once, possibly his life might have been spared. His death caused universal sorrow

among the intellectuals of the city and in Cambridge, his funeral being largely attended by prominent physicians, educators and men of affairs. He was the second person to be interred in Mount Auburn Cemetery.

As we look upon Spurzheim's cranium tonight, which Dr. Warren has so well described, we must feel that there was something in the larger aspects of phrenology, after all, for the anatomist, viewing this skull without knowledge of its origin, would surely say, "Here is the cranium of a gentleman and a scholar."

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A PLAGUE OF "HEALERS"

THE *New York Times*, most ethical of newspapers, has the following to say about irregular practitioners of medicine:

"The State Board of Regents at Albany has taken away his medical license from a well-known proprietor of a 'House of Health' in this city. The complaints about his methods were varied, and certainly seem to justify the action of the Regents. But, though this was an important case, the Secretary of the Board of Medical Examiners seems to have been unduly sanguine in predicting that it would have the effect of largely cutting down 'quackery in the medical profession.' The very 'doctor' in question had snapped his fingers at the Regents in advance, and had said that it would not make a particle of difference to him if his license were taken away. He would go on making money out of the trusting public just as before.

"It is an undeniable fact that irregular practitioners have greatly increased in number in this country, parallel with the efforts to improve medical education, to get rid of dubious schools of medicine conferring questionable degrees, and to stiffen the requirements for a medical license. In 1924 the Association of American Medical Colleges appointed a committee to make a special study of the whole question of medical education and medical practice in the United States. Last month it issued a preliminary report in the course of which it stated that 'between 1910 and 1920 there was an increase of 116 per cent. in the number of healers of various kinds other than medical practitioners and osteopaths. In all probability the numbers have continued to rise since 1920.' It is an old and curious story. Alongside science we always have ignorance raising its unabashed head. So

long as a great multitude continues to have superstitious ideas about the causes and cure of disease, so long will healers flourish in the land, whether they are or are not permitted to call themselves doctors."

EXPECTATION OF LIFE

DR. WILLIAM H. WELCH, Chairman of the Milbank Memorial Fund, which is conducting health demonstrations in three New York State centres, after stating that few of the great discoveries of preventive medicine, except in the prevention of yellow fever, are anywhere nearly fully applied, said that between 1911 and 1922 the mortality rate from all causes in the United States decreased from 14.2 to 11.8. In New York State, during the same period, it decreased from 15.7 to 13.0; and in New York City between 1910 and 1922, from 16.0 to 11.9. In the last sixty-five years the average length of life has been extended fifteen years. New discoveries, utilization of recently-acquired knowledge, closer application of accepted principles of hygiene, improved working and home conditions—these are counted on to bring still further decreases in the death-rate. But despite the spread of medical knowledge among the people, much remains to be done towards educating the general public in the ways of conserving health.—*London Medical Press Circular*.

SCARLET FEVER INCREASE

SCARLET FEVER, according to the weekly health review made public by the United States Public Health Service March 3, showed an increase of over 1,000 cases for the week ending February 12, 1927, as compared with the corresponding week of 1926. For the current surveyed week forty-one states reported a total of 5,977 cases as compared with 4,851 a year ago. Reports from 97 cities, situated in all parts of the country, and having an aggregate population of more than 30,500,000, showed the presence of 2,265 cases as compared with 1,713 for the same week of 1926. The estimated expectancy of these cities, based on the experience of the last nine years, excluding epidemics, was 1,334 cases.

Measles, on the other hand, showed a sharp decline in prevalence according to the reports of 38 State health officers. These 38 states had 11,837 cases for the surveyed week as against 17,646 cases for the week of 1926. The year 1926, however, was a measles year, so a decrease in rate should be expected for the current year. Despite the epidemic prevalence of influenza in Europe, 81 cities of the United States reported for the same week only 984 deaths from influenza and pneumonia, as compared with 1,371 deaths for the corresponding week of 1926.

**Case Records
of the
Massachusetts General Hospital**

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN
WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 13101

AN OBSCURE ANEMIA

A Russian Jewish furniture finisher forty-one years old entered August 5 complaining of breathlessness on exertion for the past month.

Seven weeks before admission he found he tired easily. At the end of his day's work he felt like sleeping, whereas formerly he had been quite active. His appetite had been very poor. He had frequent belching of gas, occasional nausea, and once vomited. He occasionally had dull epigastric pain. The symptoms increased until four weeks before admission he had to give up his work and for several weeks had been resting on a farm. He became very weak and had dyspnea and palpitation on exertion. The dyspnea increased progressively. For four weeks his skin had been very yellow. During the past week he had felt dizzy even while lying in bed, and had had not headache but a feeling that his head was heavy and large. For a week he had urinated once at night. His best weight was 150 pounds; his weight in the Out-Patient Department August 3 148 pounds.

Records of the Out-Patient Department show a visit ten years before he entered the wards. His chief complaint then was "pains all over." Examination was negative. The blood was not examined. Two days before admission he again went to the Out-Patient Department complaining of "dizzy and fainting spells."

The family history is unimportant.

For the past ten years he had had marked shooting pains over his body. Eight years before admission he had a touch of influenza. For six years he had had attacks of rheumatism in the feet, knees, back and hands. For six years he had had pyorrhea. His teeth were carious. Occasionally while working inside he had nose-bleed. He used shellac, varnish, paints and benzol in his work. He formerly had periods of dizziness. He had occasional chest pain, not severe.

Examination showed a well nourished man with a lemon yellow pallor. Mucous membranes extremely pale. Teeth decayed, many missing. Marked pyorrhea. Lungs clear. The apex impulse of the heart was in the fifth space, coinciding with the left border of dullness, 10 centimeters from midsternum, 1 centimeter outside the midclavicular line. No other enlargement to

percussion. Action regular. Sounds of poor quality. Pulmonic second sound accentuated. A harsh systolic murmur along the left sternal border. Pulses and arteries normal. Blood pressure 110/60 to 140/55. Abdomen rather full. Shifting dullness in flanks. No fluid wave. Slight tenderness in the right upper quadrant below the costal margin. Rectal examination negative. Reflexes active. Extremities hypersensitive to vibration. Fundi showed several small patches of hemorrhage. Disks somewhat indistinct. Some small white patches near the hemorrhages, — scars.

Amount of urine 33 to 80 ounces, alkaline at one of twelve examinations, neutral at one, specific gravity 1.005 to 1.022, the slightest possible trace of albumin twice, sediment showed occasional white blood corpuscles in five specimens, occasional reds in four. Renal function 50 per cent. (Two tests.) Blood examination at entrance showed 2,900 leucocytes, 21 per cent. polynuclears, 26 per cent. lymphocytes, 1 per cent. eosinophiles, 6 per cent. large mononuclears, 2 per cent. basophiles, 8 per cent. unclassified; 36 per cent. myelocyte series; hemoglobin 50 per cent., reds 1,376,000, anisocytosis, poikilocytosis, macrocytosis, stippling, polychromatophilia, platelets diminished, reticulated cells 2 per cent. Later counts are indicated in the chart. Wassermann negative. Non-protein nitrogen 33 milligrams. Icterus index 5-8. (Slight hemolysis.) Bleeding time 12 to 14 minutes. Clotting time 10-14 minutes. Clot retraction definite but poor after twenty-four hours with three-quarters volume of white clot uppermost. Consistency firm and rubbery. Gastric analysis: fasting contents, 31 cubic centimeters, turbid, colorless, acid, mucoid; free hydrochloric acid 13, total acid 28, guaiaac negative. Test meal 33 cubic centimeters, white, sweet, thick bread suspension, free hydrochloric acid 05, total acid 50, guaiaac negative. Blood culture August 13 showed streptococcus and staphylococcus.

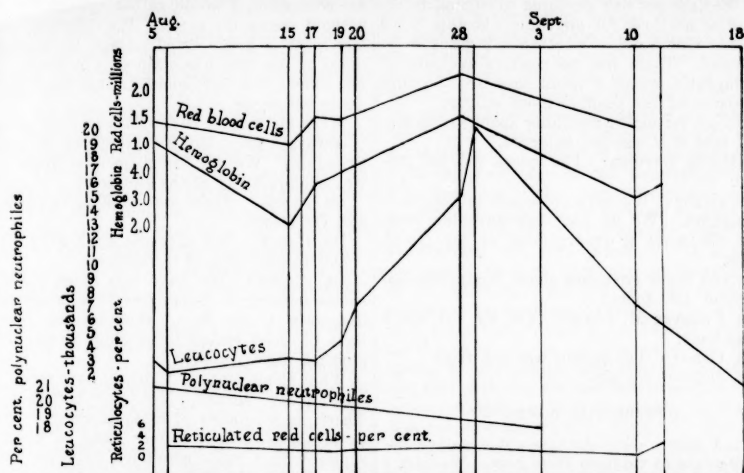
X-ray August 9. The outline of the left kidney was indistinctly seen. It seemed rather large. The right kidney was not made out. No abnormal shadows were present. Barium enema: colon appeared normal in contour. No filling defects seen to suggest organic disease. The palpable tumor mass appeared extrinsic to the transverse colon. (The clinicians found no mass.) There was nothing unusual in the appearance of the lungs or the long bones. The heart shadow was rather wide across the base in the region of the auricles. In the left lower molar region were two rounded areas of diminished density suggesting bone destruction, perhaps secondary to recent extraction, but the most anterior area was rather large and suggested partial bone destruction. September 3 there had been no marked change in the appearance of the left portion of the mandible since the previous observation. The area of diminished

density was still present. September 8 the findings were the same. There was no positive evidence of osteomyelitis.

The temperature was normal at entrance, then 99° to 105° until operation. Before operation the pulse was 80 to 120, the respiration 18 to 36.

Consultations: Oculist: Hemorrhagic retinitis, both eyes. Numerous retinal hemorrhages confined to arteries at the terminals. Hemorrhages are accompanied by small grayish-white spots. Optic discs are slightly paler than normal and

was 20,000. The temperature was 102°, the pulse 130, the respiration 53. He improved considerably and by the 26th the temperature was nearly normal, he looked much less anemic and felt stronger and the lung condition was almost cleared up. He continued in about the same condition, with some temperature. September 1 there was some infection and hemorrhage in the region of the left lower posterior molar. Two days later he felt and looked better. Blood examination however showed little improvement.



appear slightly blurred. Several red patches with white centers are present. *Dentist:* Lower left molar extracted. X-ray shows no pathology in bone. Will check up in a week with repeats to determine any advancement in possible beginning of necrosis. *Eye and Ear X-ray:* Sinuses negative. *First surgeon:* Infection of tooth socket, probably osteomyelitic, present. No treatment indicated other than oral lavage. *Second surgeon:* Advise operation.

There was much difference of medical opinion as to the diagnosis. August 14 the temperature rose to 105°, the respiration to 35. There was expiratory grunt and at the right base dullness, loud voice and breathing and moist râles. It was thought this might be a terminal event; but the following day the temperature fell to 101° and the râles disappeared. The abdomen was distended. August 16 there was definite gangrene of the cheek. That day 700 cubic centimeters of blood was transfused. Two days later the jaw looked better and the patient felt better.

It was believed that operation offered the only hope. August 20 it was done, followed by transfusion of 600 cubic centimeters of blood. Two days later there was apparently some pneumonia involving the left lower lobe. The leucocyte count

A smear showed the red cells in general well filled, though some showed achromia; moderate poikilocytosis, few macrocytes or true tiny microcytes, occasional polychromasia, an infrequent normoblast, no megaloblasts or megakaryocytes. Platelets seemed essentially normal, but were found only one to 75 red cells. There were many white cells of which the majority were leukoblasts,—i.e., very immature, abortive and sport cells. A "crude classification" was made,—18 per cent. polymorphonuclears, 36 per cent. myelocytes, 22 per cent. premyelocytes, 5 per cent. myeloblasts.

From this point the patient went downhill with increasing rapidity. On the 19th he was delirious. The leucocyte count was 1600. September 20 he became comatose. That afternoon he died.

DISCUSSION

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY AND PHYSICAL EXAMINATION

This is a set of general symptoms which with the extreme pallor and the low red count that I

see below makes one think that most of his symptoms are due to some form of anemia, whether primary or secondary one does not yet see.

You get a large pulse pressure with a great many anemias, both primary and secondary. It does not help you in diagnosis as long as there is no reason to suspect an aortic lesion.

I think we can see no reason to suspect his kidneys of anything in particular.

"Blood culture August 13 showed streptococcus and staphylococcus." Rather unexpected. We have not had anything to suggest fever. One does not look for anything like this blood culture. One wonders whether it is going to be confirmed. There was no positive evidence of osteomyelitis, which I think they are thinking of because of the positive blood culture.

I do not remember anything about his spleen, but I take it it was not enlarged.

A HOUSE OFFICER: The spleen was not palpable.

DR. CABOT: We have no reason to suspect it is enlarged. We do not hear anything more about the mass or enlargement of the lymph glands.

Do you know anything about this abdominal operation, Dr. Jones?

DR. CHESTER M. JONES: Yes, sir. It was a splenectomy.

DR. CABOT: The spleen was not felt?

DR. JONES: No, sir.

DIFFERENTIAL DIAGNOSIS

DR. CABOT: Now let us put together the definite data as we have them here. We have a positive blood culture, which apparently was not repeated. It does not look as if they thought much of it. I do not know why not. It showed both streptococcus and staphylococcus. We naturally associate that with the process in his jaw. Whether or not it is associated with that process I do not know, but they evidently were worried about that process in his jaw. The rest of the body so far as I see shows nothing to explain the positive blood culture until we get this pneumonic process, although it does not look like the main thing.

A transfusion comes in and does him good. So we have a patient who comes in with anemia, we know he has no enlargement of the spleen, and we have this sepsis, and we have nothing else. I should think this was the natural place to take up discussion as to the cause of this anemia. In the first place we naturally think of pernicious anemia. He is a middle-aged man who without any definite organic lesions such as cancer or tuberculosis or syphilis or nephritis shows a profound anemia. But he ought not to have pernicious anemia. Many of you remember the discussion just a week ago at the Brigham Hospital, where we were confronted with a very similar situation. I shall say now

as I said then that the points against pernicious anemia are the absence of any tongue symptoms, the absence of any numbness of the fingers or toes, the absence of any definite evidence of swinging up and down or any remissions, which are almost never absent with pernicious anemia, and lastly the presence of hydrochloric acid on two occasions in his gastric contents. That is strong evidence. From the blood itself, as far as the red cells are concerned, I do not believe that anyone can say. As far as the white cells are concerned, I would rather not say anything without seeing the blood. The difficulty is that one is at a great disadvantage in looking at blood unless one has stained it oneself. I do not believe it is pernicious anemia from the evidence before us.

What else can it be? Cancer of the stomach is always a good thing to think of on general principles. We get fooled on it so often. Did we have an X-ray of the stomach?

DR. JONES: There was nothing in the stomach, Dr. Cabot.

DR. CABOT: Did they get an X-ray?

DR. JONES: Not of the stomach.

DR. CABOT: We have this chest plate. It shows nothing in particular in relation to his diagnosis, I take it. I should think then we have no good reason to consider cancer of the stomach any further.

What about sepsis? Can you have sepsis with such a low count? Yes, you certainly can. There are a number of cases on record of puerperal sepsis, and not only cases of puerperal sepsis, with a white count not only low but abnormally low like this. So far as the total white count is concerned I do not see how one can exclude sepsis. The history and the picture in the rest of the case do not seem much like it. It does not seem as if he had enough evidence of sepsis to explain such an extraordinary anemia. There again we should like to know whether or not that blood culture would have been positive again if it had been repeated.

DR. JONES: The first blood culture showed no growth in one flask and streptococcus and staphylococcus in the other. A second one was taken, but we have no record of that.

A HOUSE OFFICER: It was negative.

DR. CABOT: Then I should not pay a great deal of attention to that, since one flask was negative and a second attempt got nothing. One takes sepsis as the main cause of death a little less seriously, if that is ruled out.

You all remember the case of anemia which we studied just a week ago and saw the marrow sections thrown up on the screen and called myeloblastoma. That is not yet, I think, satisfactorily named, but as far as the anemia is concerned it caused a myelophthisic form of anemia, where the marrow is pushed out, atrophied, as a result of overgrowth of some-

thing else, something else that seemed to suggest none of the lymphocyte series in that case, but rather something of the myelocyte series, so that in the end it seemed to Dr. Minot and the others who talked it over that if it was called a myeloblastoma it would be better than any of the more popular names like leukanemia or aleukemic leukemia, which are very unfortunate names. I certainly do not see any evidence to say that this case is not the same thing. That was the first case with necropsy that I have had anything to do with. Without more study of the blood I cannot say.

Can this be a splenic anemia? Not under any ordinary definition in which that term is used—with a large spleen, which is apparently not present here.

I do not know what kind of anemia it is. It may perfectly well, as far as I see, be the same kind we saw a week ago at the Brigham Hospital. There is nothing in this description of the blood that makes it clearly different from that, so far as I recollect. The only thing that I feel clear about is that it is not pernicious anemia or the ordinary type of aplastic anemia. It certainly does not correspond with that in a good many respects. That is about as far as I can go.

Another condition which I did not mention and which I cannot exclude is lymphoid leukemia with a terminal low count. I have seen a good many cases with a high white count until a terminal sepsis knocked it down, and this blood so far as I can tell by looking at one specimen for a few seconds is not unlike that. If that were true one would expect to find the bone marrow (on which the whole definite conviction here must rest) replaced by cells of the lymphocyte series.

A STUDENT: Could it have been related to his occupational history?

DR. CABOT: We do not know much about that. There are a number of substances mentioned, but without more study of the amount of benzol he inhaled or the presence of any of their derivatives in his body we cannot go any further. I am glad you mentioned it. There are some substances mentioned there that have caused intense anemia.

A STUDENT: What are your objections to its being an aplastic anemia?

DR. CABOT: In the first place there is too much evidence of regeneration. In a true aplastic anemia we do not see evidence of regeneration; but there is such evidence here. Then the vast majority of cases are in young girls, usually under thirty.

A STUDENT: In what classification do these sport cells go?

DR. CABOT: Just as you like—anywhere you feel like putting them.

A STUDENT: If it is a lymphoid leukemia how can you explain the number of myelocytes?

DR. CABOT: If I were doing that I should not say that they are myelocytes. That is of course a rash thing to do without more study of the blood.

Will you discuss this case, Dr. Jones?

DR. JONES: This is one of the most doubtful cases I have seen. It is interesting, because nobody was able to agree upon the actual findings in the blood. It was not pernicious anemia, the white cells being entirely unusual for pernicious anemia. There was free hydrochloric acid, no tongue symptoms, no jaundice, and it did not seem like a pernicious anemia clinically. The presenting feature of the case when it came in was bleeding from the mouth and mouth sepsis, and the first effort was to try to determine whether it was primary or secondary. We finally came to the conclusion that it was secondary, based upon submucous hemorrhages with subsequent infection. Those are very frequent in various types of anemia, but principally in the type that Dr. Cabot has mentioned, namely lymphatic leukemia with a rather aplastic bone marrow, a lowering of platelets, subsequent hemorrhages and then infection. It is rather common for us to see these patients come in with mouth infection. We felt that the sepsis was secondary rather than primary. The question as to the actual process going on was never decided. After a lot of discussion we finally agreed to compromise and came to the conclusion that probably the case was a so-called aleukemic leukemia, a very poor term, because it does not describe what is going on. The characteristic blood cells of which Dr. Cabot has seen a few were not easily classified. They were not typical lymphocytes and were not typical myelocytes. When we showed them to Dr. Wright he said it was absolutely impossible to classify them—simply sport cells, probably of myeloid origin, but he could not tell. As the disease progressed it was evident that there was more or less hypoplasia, with a lymphocytic or myelocytic formula, rather than a polynuclear, and there was a low white count except on certain occasions, when there was very marked increase in the sepsis and continued hemorrhages from the buccal mucous membranes. Here was a case with marked anemia, a low white count, sepsis and quite a definite amount of aplasia. The spleen was not palpable. We did not know what to do with the man. Transfusion helped him only temporarily. It stopped his hemorrhages for a very short time, a question of two or three days; then the hemorrhage would recommence. We had one case the year previous which resembled this case rather closely. The white cells were not quite the same, but there was aplasia. There was not a large spleen. The man had been transfused repeatedly with very little improvement. We finally did a splenectomy, because the man was in very bad condition and was willing to try any kind of treat-

ment that offered a chance of recovery. He developed a very good remission as far as the red cells were concerned and at present is alive—two years and some months after the operation—with a perfectly normal red count.

We also had one case that was operated upon in the hospital some seventeen years ago and which is a little like this case. The diagnosis was in doubt before operation. The patient did have a spleen that was slightly enlarged. A splenectomy was done subsequently. Within a few months after it the patient improved and at the same time developed a leucocytosis and a blood picture absolutely typical of myelogenous leukemia. She is still living, one of the few cases of this disease that have lived as long as she has following a splenectomy.

With that background, in spite of the fact that the spleen was not enlarged, our only chance of effecting any change in this case seemed to be a splenectomy. This was done. After it he did improve for a short time, the reticulated count went up and the red cells showed a definite increase, outside of transfusion. The white cells however remained abnormal throughout. He finally died from slight hemorrhage and more sepsis.

The diagnosis as far as we were concerned was never settled, but we had felt that probably we had been dealing with a case of peculiar anemia due to a so-called aleukemic leukemia.

DR. CABOT: Is there anyone else here who saw the case during life?

DR. RAPHAEL ISAACS: There were certain features in this case that pointed to chronic or subacute benzol poisoning,—the decreased number of platelets, the hemorrhages, the gastro-intestinal symptoms which are found in all cases characteristic of benzol poisoning, also a reduction in resistance to infection. The antibodies apparently are not active as normal, and patients with subacute and acute benzol poisoning are very prone to sepsis. If this man had benzol poisoning the infected teeth were a starting point for sepsis. The uric acid excretion is not increased. According to some the leucocytes are really in the inside of the body, in the capillaries of the liver and the spleen. So we probably may not have abnormal leucocyte destruction in the beginning. After a time however in benzol poisoning the bone marrow itself produces abnormal cells and may become aplastic. Under such circumstances, especially in the presence of sepsis, it is possible that the bone marrow would make every effort to produce new cells. We found abnormal cells. We should probably find abnormal cells in different organs from the myelocytic series. The other symptoms are those of anemia and symptoms which may be present in chronic benzol poisoning with sepsis and secondary reaction on the part of the bone marrow.

A STUDENT: Was there evidence that he had close contact with benzol?

DR. CABOT: It is mentioned, but it does not say how close.

DR. JONES: We went into his industrial history. He was a furniture polisher and had used benzol from time to time. None of the other men on the same job had any benzol poisoning symptomatically.

DR. CABOT: It seems to me that that is an essential link in your chain. Benzol is a substance a good many people use and do not have any trouble with. You have to say that he is using more of it or in some way he gets more in his system than another person. It might be a special idiosyncrasy or sensitiveness, but that is rather dangerous ground, I think, to take.

The whole interest here is on what the marrow is going to show. I have gone on record and so has Dr. Jones that it will not show the type of marrow associated with pernicious anemia. Dr. Jones thinks it will show, on the contrary, some aplastic anemia. I do not feel so certain about that. I have not studied the case so much as he has. If it were a benzol poisoning it should show in the marrow chiefly loss and not increase of cells, but I should say that that is not probable. We ought to see increase of cells, on the whole of the myelocyte type, not of the lymphocyte type. If it was on the other hand a lymphoid leukemia, with the white count falling suddenly at the end of life, then the marrow ought to show increase of cells of the lymphoid type. These are the possibilities on the two sides. I think if I had to go without any more evidence than I have I should favor the last of these hypotheses.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Leukemia, myeloid.
Stomatitis, ulcerative.
Splenectomy for aplastic anemia.

DR. RICHARD C. CABOT'S DIAGNOSIS

Lymphoid leukemia?

ANATOMICAL DIAGNOSIS

Aplastic anemia.
Atrophy of the bone marrow.

DR. MALLORY: The bone marrow was the only thing in the case that we were allowed to examine. Much to everyone's surprise it was an extremely atrophic bone marrow. In gross it was largely fatty, but with a slightly more gelatinous consistency than perfectly normal fatty marrow. It was not red, as it would be in pernicious anemia, and it was not white, as you would expect it to be in a leukemic process. Very few cells were present in the microscopic sections. Of the ones identifiable there were a considerable number of eosinophils, very few myelocytes, very few normoblasts and practi-

cally no megakaryocytes found. A great deal of the fat tissue was replaced with a very fine fibrillary network apparently at least resembling coagulation serum. The whole picture is fairly characteristic of what sometimes is called a serous atrophy of the bone marrow. So far as I know anything about benzol poisoning that is consistent with it, but by no means diagnostic.

DR. CABOT: Between that and aplastic anemia is the general impression you get from the marrow?

DR. MALLORY: Yes.

DR. CABOT: It seems to me that for practical purposes the importance of this case and the one last week is that they are not benefited by transfusions, and we cannot hope to do them any good through the feeding of liver.

A HOUSE OFFICER: This man was fed liver.

DR. CABOT: I will read the pathological report on the spleen, which is the only other fact we have on his morbid anatomy. "Spleen 11x8x3.5 cm. On section firm, dark red, with normal follicles. Microscopic examination shows the natural structure obscured by a diffuse and dense infiltration by cells most of which seem to be of the nature of myeloblasts and erythroblasts. Some megakaryocytes and many cells containing blood pigment are present.—Myeloid hyperplasia."

DR. MALLORY: Dr. Wright also saw the bone marrow. We were unable to bring the two together at all.

DR. CABOT: You can take your choice. It seems to me that for practical purposes the diagnosis of cases like this is now between (a) the intense anemia that will be benefited by liver, (b) intense anemias which we have no reason to suppose will be benefited by liver and (c) those benefited by splenectomy. This man had the benefit of both and did not get better.

A STUDENT: Do you think the operation was justified?

DR. CABOT: Here is a man on whom we know nothing definite, whose diagnosis in spite of a great deal of study cannot be settled, and we are willing to take any chance. Yes, perfectly justified.

DR. JONES: He did not die from the splenectomy at all. He died from the disease.

A STUDENT: How about the high color index? Is not that unusual?

DR. CABOT: Yes. This whole case is unusual. Everything about it is unusual. I do not know that any of us will ever see a case just like it. But you can say certainly that it is unusual to have any high color index which is not pernicious anemia. I think it is well to remember that he did not have tongue symptoms, did not have jaundice and did have hydrochloric acid.

A STUDENT: With the bone marrow picture given is it possible that that is lymphatic leukemia?

DR. CABOT: No. I think that diagnosis is

impossible in view of what Dr. Mallory has said.

A STUDENT: Then what conclusion can you come to?

DR. CABOT: That it can be explained as benzol poisoning or aplastic anemia of unknown cause, so far as the marrow goes. It seems to me that aplastic anemia is the best diagnosis. Is that what you think, Dr. Jones?

DR. JONES: It is not idiopathic aplastic anemia of the usual type. It is very unusual. It is not the typical picture, with red cells simply small and pretty well shaped and lymphocytes and no abnormal white cells.

A STUDENT: Is not the clinical picture quite typical of benzol poisoning?

DR. CABOT: I should say an essential part of the clinical picture of that disease is the knowledge of an unusual absorption of benzol. That we have not got.

A STUDENT: Is not the only thing against the poisoning the simple fact that you have not got that evidence? It does not exclude the diagnosis.

DR. CABOT: No. I think anyone has a perfect right to believe it is that.

A STUDENT: Are there other cases of benzol poisoning showing pictures like this?

DR. CABOT: Not so far as I know.

DR. JONES: Dr. Minot was given all the details of the case, and he thought it was not characteristic of benzol poisoning. That was before we had seen the marrow.

CASE 13102

A PULMONARY SUPPURATIVE LESION. REMARKS ON DIAGNOSIS AND TREATMENT

MEDICAL DEPARTMENT

Man, 31, married, born and living in Massachusetts. Clerk in a meat-market.

Past history. Always well with the exception of measles.

Family history. Negative. No tuberculosis or exposure. Married ten years. No children. Wife has had no miscarriages.

Habits. Good.

Present illness. Seven months ago his tonsils were removed. One week after the operation he began to cough, raising purulent green or yellow sputum, occasionally streaked with blood. No frank bleeding. No pain at the onset of pulmonary symptoms. He has continued to cough since about three to four times every hour day and night. The cough is paroxysmal. At first the cough was more troublesome in the morning and on lying down at night and he raised two to three ounces of sputum at each paroxysm. The constantly recurring paroxysms of cough would result in the expectoration of nearly a pint of pus in about an hour. At times

he vomited and brought up a large quantity of vomitus and sputum. Two weeks ago the cough stopped for two days. This was followed by an attack of fever with a temperature of 100° to 103° for a half a day. He then coughed up about one half pint of foul sputum within a period of about ten to fifteen minutes. The paroxysm of cough is brought on by talking. The cough is improving. He coughs less often and raises less sputum. Five and six months ago he raised about a pint in 24 hours. He now raises from one-half to one pint. The breath is foul. The sputum is not foul.

Immediately following the operation the temperature was elevated, 100° to 101° for three weeks. It then became normal and he went to work. He has since had attacks of fever coming every three to four weeks and lasting ten to fifteen hours. The temperature is usually 100° to 101° , rarely reaching 103° . Preceding the febrile attacks he coughs less for five or six hours. During the course of his illness he has had many chills and sweats, none however for three months.

Pain during the illness in the right side of the chest after cough, not increased by long breath. Appetite poor. At times attacks of nausea and vomiting; for one month four months ago every day, less often since then. No distress after eating. No gas. Bowels move every day. Average weight about 125. He has lost about fifteen pounds during the present illness. He does not get up at night to pass urine.

Physical examination. Well-developed and fairly nourished. Slight exophthalmos. Puffiness of the lower lids. Pupils equal and react to light. Gums show some pyorrhea. Throat negative. Tonsils absent. Glands not enlarged. Breath slightly foul and very foul after a paroxysm of cough. Heart normal position, not enlarged, negative.

Lungs:—Slight depression of the right front of the thorax and slightly diminished respiratory excursion of the right lung. The right front is slightly dull throughout. In the first and second interspaces from the sternal margin to a hand's breadth outside there is slightly greater dullness. Behind there is slight dullness between the vertebral border of the scapula and the dorsal spine over an area extending from the level of the angle of the scapula to within two finger-breadths of the spine of the scapula. Respiration is slightly diminished throughout the right side. An occasional coarse sonorous râle is heard after cough over the right upper lobe. Above the midscapula behind the expiration has a sibilant quality. No bronchial breathing and no moist râles. Examination of lungs otherwise negative. No cracked pot sound on percussion.

Abdomen negative with the exception of a marked impulse on cough in the right internal

inguinal ring. Genitals negative. Blood pressure 120/70.

Sputum mucopurulent, without blood. Examination shows numerous organisms resembling pneumococci and some influenza bacilli. No tubercle bacilli in seven specimens. No elastic tissue.

Blood:—Whites 9,000. Hemoglobin 75%. Differential count negative.

Stools negative. Urine shows nothing abnormal.

Temperature during one week of observation normal or subnormal in the morning and usually 99.2° in the evening. Pulse about 80. Respiration 20.

X-ray shows marked thickening about right lung root. Area between 2nd and 3rd ribs in front rather extensive mottled dullness. Apices and bases clear.

2nd X-ray, two days later. In the center of dull area some diminished density above with increased density below, nearer front and in lateral view 6 centimeters in front of spine and nearly opposite the shadow of aorta. In antero-posterior view about 6 centimeters to right of median line and in the 2nd interspace; behind between 6th and 7th ribs. The increased density in the lower part of the involved region has a horizontal upper margin suggesting a fluid level.

DISCUSSION

BY FREDERICK T. LORD, M.D.

The history indicates a pulmonary disturbance after a tonsillectomy. The foul sputum and expectoration of large amounts of sputum in a short period suggest a pulmonary abscess or empyema breaking into the bronchi. The absence of pain in the early part of the illness is in favor of lung abscess rather than empyema, but pain during the course of the illness suggests intercurrent involvement of the pleura. The cessation of cough and coincident febrile reaction suggest an imperfectly evacuated pocket of pus. It is not uncommon at times in such cases to have the breath foul and the sputum not foul, and this may mean that the expectorated material does not come from the abscess.

NOTES ON THE PHYSICAL EXAMINATION

The process is evidently right-sided and chiefly in the upper part of the chest. There are no signs of cavity, but this is of no moment in excluding cavity, as losses of pulmonary substance can seldom be demonstrated in non-tuberculous cases on physical examination.

It is to be noted that no cavity is described in the first X-ray, but that in the second X-ray there is an area of increased density with central increased radiance below which is density with horizontal upper border suggesting fluid in a cavity. The absence of any such appear-

ance in the first X-ray suggests that the first film was taken with the patient prone or supine. This is unfortunately a common mistake and responsible for many failures to demonstrate collections of air or fluid in the pleural sac or in the lung. Such failures can be avoided by taking an anteroposterior film with the patient erect, or if this is impossible with the patient lying on his side. With the patient lying on his side the film is placed on the back or front of the chest. Cavities with air and fluid may also be demonstrated if one X-ray is taken when the cavity is full and another when the cavity is empty.

CRITICISM OF THE RECORD

There is little to criticize in the history, which gives the evolution and grouping of symptoms, and little to say of the physical examination. The Wassermann test is lacking, and it is to be noted that no mention is made of a search for spirochetes in the sputum. As this case came under observation eight years ago, this may explain the absence of the search for spirochetes in the sputum. Criticism can be made, as already noted, of the X-ray, if the first was taken with the patient prone or supine. It is also to be noted that nothing is said of bronchoscopy as one of the special examinations in this case.

REMARKS ON THE DIAGNOSTIC VALUE OF BRONCHOSCOPY AND LIPIDOL INJECTION

Bronchoscopy is of value in detecting the presence of bronchostenosis, which may be due to cicatricial fibrous tissue, to granulation tissue, to malignant disease, or to a foreign body. In this problem the onset of symptoms following tonsillectomy excludes the likelihood of foreign body and malignant disease with reasonable certainty. Bronchial plugging from the presence of granulation tissue is however a possibility.

With respect to the question of bronchial plugging it may be said that there are usually physical signs in such cases which suggest its presence. The signs differ whether the plugging is complete or partial and valvular. If the bronchial plugging is complete there are over a circumscribed area such signs as dullness, diminished or absent breathing, diminished voice, whisper, and diminished or absent tactile fremitus. The signs in this case do not suggest bronchial plugging, and the X-ray findings are also against it. In the presence of complete plugging of the bronchus the X-ray is likely to show collapse of that part of the chest wall overlying the collapsed area, with narrowing of the interspaces and a homogeneous dense shadow in the lung. The heart and mediastinum are also likely to be displaced toward the affected side.

In the presence of a valvular closure of the bronchus there is likely to be an acute pulmonary inflation over the parts of the lung supplied by the partly occluded bronchus. On physical ex-

amination such an area may show tympany on percussion with diminished breathing, voice, whisper, and tactile fremitus, and X-ray is likely to show that the affected region is more translucent than other parts of the chest, with displacement of the mediastinum to the opposite side, depression and flattening of the diaphragm and immobility of the diaphragm on that side. In this case, if there is bronchial plugging, as seems unlikely, it affects the bronchus leading to the upper lobe, and this bronchus is not readily accessible with the bronchoscope. Even if accessible relief by this means is doubtful, and as the nature and site of the process can be established without bronchoscopy there seems no necessity for its use in this case. The procedure is somewhat of an ordeal and not to be undertaken without justification.

Lipiodol injection is of service in the diagnosis of bronchiectasis, but there is no reason especially to suspect bronchiectasis in this case. In our experience lipiodol has not proved of material assistance in the diagnosis of abscess. Abscess cavities usually communicate so imperfectly with the bronchi that the injected substance outlines the bronchi surrounding the abscess, leaving the involved region itself as a blank, and the diagnosis is then made by exclusion. On the whole this means of localization has not proved very helpful, and there is no indication in this problem for its use.

DIFFERENTIAL DIAGNOSIS

Tuberculosis should be considered in the diagnosis of all pulmonary lesions, and more particularly when as in this case the upper lobe is involved. But there is usually no difficulty in making the distinction between tuberculous and non-tuberculous processes. In this patient the onset after tonsillectomy, the foul character of the expectoration, the absence of tubercle bacilli in repeated examinations of an abundant purulent sputum, and the absence of moist râles over the involved area are against tuberculosis. The presence by X-ray of a cavity, probably in the right upper lobe, and mottled increase of density above the anterior portion of the third rib is consistent with tuberculosis. There is some evidence against tuberculosis in the exclusive involvement of one side as in this case. In one hundred and eight successive cases of pulmonary tuberculosis in this hospital with positive sputum and X-ray examination eight were in the first, forty-four in the second and fifty-six in the third stage of the disease. The process was bilateral by X-ray examination in 83 (76.9%) and unilateral in 25 (23.1%). Of the twenty-five unilateral cases two were in the first, ten in the second and thirteen in the third stage. The frequency of unilateral involvement even in the more advanced types of tuberculosis by X-ray examination makes it undesirable to lay any special stress on this point in deciding the tuber-

culous or non-tuberculous character of a pulmonary process in any individual problem. In spite of the X-ray appearance here, which is consistent with tuberculosis, a non-tuberculous lesion is the most likely explanation of his symptoms.

CONCLUSIONS REGARDING THE DIAGNOSIS

We may conclude that he has a non-tuberculous suppurative lesion,—an abscess in the right upper lobe.

TREATMENT

Various procedures are to be considered: postural drainage, bronchoscopy, intravenous arsphenamin, artificial pneumothorax, incision and drainage.

In our experience, under rest in bed and postural drainage alone eleven per cent. of 227 cases of pulmonary abscess recovered. It is evident from analysis of these cases, however, that the time element is an important factor in the outcome. Thus, of 106 cases of pulmonary abscess in our series, with a previous duration of eight weeks or less, twenty-four, or twenty-two per cent., recovered under rest in bed and postural drainage. The lesions which recover under these measures are relatively mild and of short duration,—eight weeks or less previous to the institution of such treatment. The location of the abscess is important for spontaneous recovery, and in seventy-five per cent. of the cases in this group of recovered cases the process was at or above the level of the lung root, drainage into the bronchi doubtless being favored by this site. In this case a previous duration of seven months makes it highly improbable that rest and postural drainage alone will succeed in relieving the condition.

Bronchoscopy as a means of treatment is to be rejected on grounds previously noted. Treatment as for a spirochetal infection is still *sub judice* and hardly likely to be successful in a chronic case.

Artificial pneumothorax in treatment is to be entertained especially in the more acute centrally placed abscesses communicating with the bronchi and without pleural involvement. Here in the case under discussion the duration is too long, the abscess is accessible to more radical intervention, and the pleural cavity is probably involved.

Incision and drainage seemed the procedure of election in this case.

OPERATION

The patient was operated upon by Dr. Wyman Whittemore in a two-stage operation, in the first stage of which there was a resection of the anterior portion of the right third rib, with insertion of sterile gauze and closure of the wound. Three days later Dr. Whittemore explored through the adherent pleura and resected the

second rib, finding and draining an abscess below the second interspace.

The sinus continued to discharge for a long time. A letter from this patient eight months after operation states that the sinus had constantly drained, no longer than four days having gone by without a discharge, and that a small sinus about the size of a pinhead was still present. Nine months after operation a small sequestrum was discharged. Fifteen months after operation the sinus had healed.

A report from this patient nearly six years after the operation states that he has remained perfectly well, without cough, expectoration, or other symptoms.

Dr. Camp, I made the supposition, from the difference in the description of the two X-rays taken at the interval of two days apart, that because they did not describe anything suggestive of a cavity in the first, and because they did describe such an appearance in the second, the first might have been taken in an improper position to show the cavity.

DR. JOHN D. CAMP: That might be true, or the cavity might have been full. Oftentimes if we can X-ray these patients after they have been on postural drainage we can show the cavity much better than when it is full.

DR. MONROE A. McIVER: In regard to the high percentage of unilateral tuberculosis shown by cases in this hospital, I should like to ask Dr. Lord if he thinks that percentage would hold in a large sanatorium for tuberculosis.

DR. LORD: Of course it can be said that the more advanced the case the more likely there is to be bilateral involvement. I do not believe I can answer your question as to the disparity that there might be between findings in this group and any other group.

DIAGNOSIS

Abscess of the lung.

CASE 13103

A PULMONARY COMPLICATION FOLLOWING EXTRACTION OF TEETH

SURGICAL DEPARTMENT

An English mill operative forty-two years old entered the hospital September 2.

Eight weeks before admission he had some teeth extracted. Afterwards he had pleurisy. At the end of a week a tap gave clear straw colored fluid. A few days later a second tap was dry. For six weeks he had steadily lost weight.

No family history or past history was obtained.

Examination showed a very thin man with evidence of much loss of weight. There was pyorrhea. At the base of the right lung and in the maxillary region there was flatness with

diminished breath sounds and decreased tactile fremitus. The expansion of the chest on the right was limited. The whole picture was one of fluid at the right base. The rest of the examination was negative.

Before operation the urine and blood were not recorded; the temperature was 102.7°, the pulse 147, the respiration 30.

X-ray showed dullness of the entire right chest, most marked toward the base, where it obliterated the outline of the diaphragm and the right border of the heart. The heart was displaced toward the right. There was rather fine dense mottling in the upper part of both lung fields which had something the appearance of lipiodol in the lung.

Operation was done the evening of admission, and next day transfusion of 600 cubic centimeters of blood. There was copious drainage of very foul pus. The patient remained in shock. He was in much better condition after the transfusion, but showed jaundice. The urine showed the slightest possible trace of albumen, 2 per cent. sugar, specific gravity 1.020, a few brown granular and hyalin casts. A medical consultant found no evidence of cardiac displacement or pathology in the left lung. "The patient needs 4000-5000 cubic centimeters of fluid daily. The sugar in urine may be an incident. Do blood sugar." September 22 he was worse again. The temperature was normal, but the pulse was 122 to 140, the respiration 29 to 38. September 23 he was better in the morning. Another 600 cubic centimeters of blood was transfused. He failed rapidly during the day. The jaundice was largely gone. September 24 he died.

DISCUSSION

BY MONROE A. MC IVER, M.D.

Pulmonary complications coming on soon after an operation on the upper respiratory tract, especially when this operation is done under general anesthesia, at once suggest the possibility of a lung abscess. We do not know whether this man had his teeth extracted under ether or not. Afterwards he had a pleurisy, and at the end of a week a tap gave a clear straw-colored fluid. I should say that if that history is accurate it would not point to a lung abscess.

The rest of the history is so meagre that it is very difficult to draw any conclusions from it. All we know is that the patient is said to be losing weight. Other things that we should obviously like to know are the amount of fever, if any, that he has had, whether he has had any night sweats, whether he has had a cough, and if so the amount and character of the sputum. We should also like to know whether he has been running a high leucocyte count.

The physical examination that is given to us

is certainly very characteristic of fluid in the chest, and this is confirmed by the X-ray plate, which shows marked density over the right side of the chest, especially over the lower part of the right side. We can see some lung markings towards the apex.

As I said, it seems to me from the physical examination, taken in connection with the X-ray plate, that the diagnosis of fluid is obvious. The next question which naturally occurs to us is, with what type of fluid are we dealing? Presumably after the diagnosis of empyema had been made and before the operation was undertaken a needle was put in in order to confirm the diagnosis and also to gain as much data as possible in regard to the character of the fluid. It might have been dangerous to wait for reports of culture on this fluid; but a great deal could have been learned by a macroscopic examination of the fluid supplemented by a stained specimen.

X-RAY INTERPRETATION

Evidence of an extensive process on the right side involving both the lung and the pleura.

DR. MC IVER'S PRE-OPERATIVE DIAGNOSIS

Empyema.

PRE-OPERATIVE DIAGNOSIS

Right empyema.

OPERATION

Under local novocain a two-inch incision was made in the posterior axillary line parallel to the eighth rib, and dissection carried down through fascia and muscle to the rib. The periosteum was opened and stripped from the rib for a distance of about an inch and a half, and about an inch of rib was removed. The pleura was infiltrated with novocain and dissection carried down through the pleura with a knife into the thoracic cavity, where thick foul pus was obtained; but no quantity of it was allowed to escape at the time of operation. A large soft rubber tube and a Dakin tube was placed within the pleural cavity and sutured in place. The skin laceration was approximated by several silkworm-gut sutures.

FURTHER DISCUSSION

It is suggestive from the pulse rate of 147 that this patient was very sick before operation. I do not see much else that would indicate this, as the history and physical examination are given.

It seems to me desirable that this operation should have been carried out under local anesthesia, as it was. It is a simple matter to remove a section of a rib under local anesthesia, with little discomfort to a patient, and the shock

of the operation is much less than if the patient is subjected to a general anesthesia.

Another procedure that we are likely to use if the patient is quite sick is the so-called catheter thoracotomy. In this operation a trocar is introduced into the thoracic cavity and a catheter is slipped in without removing any portion of rib. This also can easily be done under local anesthesia. There is no shock from the operation, and it causes less violent pressure changes in the thorax than taking out a rib and making a large opening into the pleural cavity.

If the lung is adherent around a walled-off cavity a large opening makes little difference. On the other hand if the lung is not adherent, and this is unlikely in the type of empyema caused by the streptococcus, the lung on the affected side may collapse when the chest cavity is opened, and the patient may suffer a considerable amount of shock.

It is noted in the operative record that a great deal of pus was not drawn off immediately at the time of operation, but was presumably removed during the twelve hours succeeding the operation. This is a good procedure because it allows the thoracic viscera to become adjusted to the new conditions of pressure. If we remove a large amount of pus at one time it may disturb the equilibrium.

The statement is made that there were two transfusions the day after the operation, and the patient remained in shock. I do not believe that this is a shock due to operation, the so-called surgical shock. I think it much more likely that it is due to a profound toxic condition, and under those conditions the results of transfusions are usually disappointing. We do use blood transfusion in certain cases of sepsis that we encounter in surgery, but this is usually with a view of combatting secondary anemia rather than with any hope of lessening the toxemia.

The jaundice noted was apparently transitory. I take it to be a hemolytic type of jaundice, and probably in itself not of a great deal of significance.

The fact that although the temperature came down to normal the pulse rate still remained very high I should say is a rather poor prognostic sign. We would much rather have seen the temperature stay up and the pulse rate tend to come down.

Some of the other data that we would like to have in this case would be, in the first place, the character of the pus obtained at the time of operation, and the type of organism obtained on culture. I think it would also be very interesting to know if a blood culture was taken, and whether that was positive or not.

MISS PAINTER: None is recorded.

DR. McIVER: I should think that there was probably other sepsis present. The empyema was drained, and so far as the record shows it

was without any favorable influence on his condition.

DR. FREDERICK T. LORD: If the pus were foul would you advocate operation anyhow, without of necessity waiting for examination of the pus?

DR. McIVER: I do not think it would be necessary to wait. It would take only a few minutes to make a smear. I think it would be interesting, but I do not believe it would be necessary.

DR. JOHN D. CAMP: In this film the left lung field shows multiple very small areas of increased density which undoubtedly represent areas of calcification. It is the type of thing which has been called healed miliary tuberculosis, and what the relation of this process is to that on the other side I would not like to say.

DR. LORD: Is there anything else that can give that appearance?

DR. CAMP: There is only one other thing that will do that, and that is in some of these cases that have had lipiodol put into them. Lipiodol gets into the small alveoli and gives that appearance. I do not know any reason for this patient to have had lipiodol. The areas are too small and too dense for a metastatic area. It is what we call healed miliary tuberculosis.

It is interesting that in spite of fluid at the right base his heart does not seem to be displaced, and I think that means that there is some process holding it over, either adhesions, new growth or a partially collapsed lung. We frequently see this displacement of the heart towards the side that contains fluid in new growth of the lung. I do not believe this X-ray will allow us to make a differential diagnosis, but we can go so far as to say that there is fluid in the right base, something holding the heart over, and healed miliary tuberculosis.

DR. LORD: I may say that there is one case which came to necropsy at this hospital in which there was evidence of healed miliary tuberculosis with calcified lesions sparsely scattered through the lungs, obsolete disseminated tuberculosis of the liver and the spleen and a calcified bronchial lymph gland. There was also a calcified area in the left ventricle, and this may have been the source of the miliary tubercles from which the patient had recovered.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Empyema of the pleura following pneumonia.
Septicemia.
Thoracotomy.

DR. MONROE McIVER'S DIAGNOSIS

Empyema of the pleura.
Other sepsis.

ANATOMICAL DIAGNOSIS

1. Primary or fatal lesions.
Empyema.
Gangrene of the lung.
Pneumoconiosis.

2. Secondary or terminal lesions.

General peritonitis.
Acute periappendicitis.

DR. MALLORY: The lungs here presented an interesting picture. The entire pleural surface of both lungs was studded with little white granulations each of which had a black anthracotic border. The lesions did not seem to be calcified, but were extremely dense, fibrous, and showed histologically a slight amount of unidentified crystallized material in the center of each, surrounded by a wall of very dense fibrous tissue, and peripheral to that a wide zone of infiltration by endothelial leucocytes filled with carbon. There was nothing in any of them on which we could possibly make a positive diagnosis of miliary tuberculosis. The lesions were present also throughout the lung parenchyma, not merely subpleural. We had another case today that had similar subpleural lesions, but none elsewhere in the lung.

Against its being a healed miliary tuberculosis I think is the fact that no foci were found anywhere else in the body. I should certainly have expected to pick up some in the liver. If it is not that I do not know what we are going to call it. Possibly it is an inhalation phenomenon, but it certainly is not the usual picture.

As to the facts of more immediate significance regarding his death: the right pleural cavity was almost completely obliterated with dense fibrous adhesions, and at the region of the operative wound was a small pocket of greenish pus. The lung was tightly adherent around that, and was so necrotic and friable that it was impossible to separate it from the pleura without tearing it pretty much to pieces. There was, however, nothing that suggested an abscess. I think it is probable that a pleurisy occurred first which secondarily developed into an empyema, rather than that the case is one of lung abscess.

DR. McIVER predicted correctly that we should probably find some other form of sepsis beyond that in the pleural cavity. There was a general peritonitis with the entire abdomen filled with fibrin and purulent exudate. The remainder of the necropsy was essentially negative.

DR. LORD: There were no tubercles in these areas?

DR. MALLORY: No, and moreover he had no tuberculous glands. That is another point that I think is strongly against miliary tuberculosis. I cannot imagine miliary tuberculosis of that extent that would not have drained into the bronchial glands.

DR. LORD: In adult types of tuberculosis are the neighboring glands involved?

DR. MALLORY: Very frequently, I think.

DR. LORD: I thought that that was the point which Ranke made, and that in his primary com-

plex initial pulmonary tuberculous lesions give rise to regional glandular involvement, but that secondary tuberculous lesions in adult life do not ordinarily give rise to neighborhood glandular enlargement.

DR. MALLORY: I should rather disagree with that so far as my own experience is concerned. For instance, I have seen a case in which one could demonstrate a calcified primary focus in the lower lobe, with a calcified satellite gland, then a secondary focus in the apex, and more tuberculous glands in the region draining the apex which were not yet calcified. Finally tuberculous enteritis, probably a third stage, and tuberculosis of the mesenteric glands, probably most recent of all. Such three-stage processes are perhaps unusual, but I am sure the two-stage is not uncommon.

DR. McIVER: I should like to ask Dr. Mallory if he thinks the peritonitis was a terminal infection or started there and spread above the diaphragm?

DR. MALLORY: I think it is terminal. The process in the pleura was certainly an earlier one than the process in the peritoneum. I cannot make an exact estimate of the duration of the latter, but think it was not more than four or five days.

DR. McIVER: Sometimes in empyema we can get a suggestion of trouble below the diaphragm by the characteristic odor of colon bacillus infection from the pus removed from the chest. When the organism responsible for the empyema is the colon bacillus the original infection is often found in the abdominal cavity.

DR. LORD: Do I understand that all these areas were pleural?

DR. MALLORY: No; they were both pleural and pulmonary.

HOSPITAL CLINICAL CONGRESS

MARQUETTE University College of Hospital Administration has announced plans for the holding of the Hospital Clinical Congress of North America the week of June 20 to 24 inclusive, in Milwaukee.

There will be four distinct departments: Hospital, Public Health, Safety and Research. The Congress will be vitally interesting to all persons interested in hospitalization; hospital superintendents, staffs, trustees, nurse superintendents, engineers, architects, dietitians, those interested in safety, first aid industrial hospitalization; public health leaders, welfare workers, both public and private.

The detailed program for the Clinical Congress will be completed within a short time and ready for publication. Information on the short and summer courses can be secured from Dean John R. Hughes.

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THE MENACE OF INFLUENZA

REPORTS in the daily press and from the United States Public Health Service emphasize the need for precautions against influenza, which is now occurring in epidemic form throughout most of Europe. In spite of all the studies made during the great epidemic of 1918 and since that time, we know relatively little more about the epidemiology of this disease and so our efforts at prevention and control have at present only an empirical basis. A good many investigators, however, are gradually swinging to the belief that Pfeiffer's bacillus is the etiological factor, thus tending to confirm much of the work done in the 1918 epidemic. However, this is far from proved at the present time.

Although we ordinarily consider Asia or eastern Europe to be the sources of supply for our various epidemic diseases, it is rather interesting that Spain has appeared to be the starting point for both the 1918 epidemic and for the present European outbreak. Whether this point will prove to be any help in the study of the disease is yet to be seen, but at least it is well to remember that our Western civiliza-

tion can not always protect itself from disease by guarding its eastern portals.

Fortunately, as yet the United States has escaped influenza in anything like epidemic form, although the past few weeks the prevalence of mild febrile conditions combined with symptoms of upper respiratory disease has become increasingly noticeable.

It is well for us all to be on our guard against respiratory infections, however mild they may appear, and to avoid so far as possible transmitting them to or acquiring them from others. Whatever we may ultimately find to be the cause of influenza, it is fairly safe to say that it will run true to the other infectious diseases in increasing its virulence with rapid passage from one person to another. Therefore the most effective way in which we can help the quarantine authorities in preventing the outbreak of influenza in epidemic form in this country is to insist on a serious attitude toward even the mild respiratory infections and to urge that every care be taken to avoid transmitting such an infection to others.

AGAIN THE HERALD PRESCRIBES

THE Americans' bill for patent medicines, first cost only, is a colossal sum. The patent medicine industry in this country is in the "Big Business" class and it is there because of newspaper advertising. The majority of our reputable public prints—those with a fine regard for the welfare of the public, instead of a shrewd solicitude for the prosperity of their shareholders—bar their columns to the nostrum vendor. Not so the *Boston Herald*. In its issue bearing the portrait of the Father of our Country, the *Herald* poses as internist, dermatologist, beauty doctor and gynecologist, and all this quite apart from, and, it might be said, quite contrary to the advice in its "How to Keep Well" column.

On one page it tells us two sure ways for preventing colds and coughs. The reader is permitted his choice of Bromo-Quinine or Creomulsin, and will probably choose the former because of its greater display. On another page the *Herald* tells you to "cheek colds in this quick way," which is Cascara Quinine. If the cold is now no better, or if it is worse, the *Herald* suggests treating colds "2 ways at once" with Vaporub. If it is a cough, another page assures you that you can "cheek it before inflammation reaches the danger zone" with Pertussin. If all these fail there is still 666, but little recommendation goes with this prescription—it pays a minimum for space.

The *Herald* would have its readers beautiful. If you are "discouraged,"—and they make the subtle suggestion of a mirror—there is Resinol Soap and Ointment, but if the face is covered with pimples, Cuticura heals. Most pulling of

all, however is the "Quick Magic of Calcium"—you get "a beautiful complexion *this week!*" "A skin that *sparkles!*"

Then there is that unfashionable fat. There is an easy way of reducing—three Marmola tablets daily will do it, and the *Herald* urges, "Prove this for your own sake. Do it now."

The internist *Herald* with a delicate reticence tells you that "you need 100% bran!" But, later because of half tones, testimonials and space, all reticence is laid aside and the *Herald* boldly tells you to "wash away constipation," and tells its costive readers that "that is Nature's way—the way of Pluto Water."

It is to be deprecated that the *Herald* shows little reticence when it warns of "Woman's Danger." It has always seemed to the editor, that the difficulties and embarrassments of lunar cycles might well be left to other agencies of information than the newspaper. The *Herald* thinks otherwise.

While His Excellency, Governor Fuller, stirred by the lamentable low standards regulating the practice of medicine in this Commonwealth, urges a high elevation of these standards; while physicians, graduates of some of the inferior medical colleges advise the revocation of these colleges' charters, and while every effort is being made to protect the public from self-dosing and wrong dosing from the quacks, the incompetent and the unscrupulous—the *Herald* prescribes.

THE HEARING ON THE GOVERNOR'S RECOMMENDATIONS FOR FURTHER POWERS FOR THE BOARD OF REGIS- TRATION IN MEDICINE

In his annual message to the Legislature Governor Fuller recommended that the Board of Registration in Medicine be given the authority to examine medical schools for the purpose of determining the eligibility of the graduates of such medical schools for examinations conducted by this Board.

A hearing on this recommendation was held by the Committee on Public Health February 23. In support of this recommendation Drs. Bowers, Stone, Vaughan, Prior, Cowles, Mr. Kiernan, and others spoke in support of the recommendations.

The opposition was led by Dr. Turner, Registrar of the College of Physicians and Surgeons of Boston. His arguments were supported by several other speakers. Dr. Turner's contention was that the Board of Registration is doing good enough work under the present law, that this movement is inspired by the American Medical Association or the Medical Trust, and that the statistics taken from the records of the Board of Registration are not interpreted correctly. He asserted that Dr. Simmons, formerly Secretary and now Emeritus Secretary of the

A. M. A., was guilty of criminal practice according to testimony which Dr. Turner could produce, that a former President of the Massachusetts Medical Society had been in the employ of the A. M. A. to further its policies in this state and that the A. M. A. has control of the activities of those who are trying to have the State of Massachusetts raise the standards of medical education and licensure, and further that those in office who may not act in harmony with the policies of the A. M. A. are in danger of being disciplined.

His harangue was particularly replete with invective and sarcasm.

The matter is now in the hands of the Committee, some of whom seemed to endorse the vaporings of Dr. Turner. We hope that there are enough members of the legislature who can appreciate the importance of providing the people with well trained physicians.

IMMEDIATE MICROSCOPIC DIAGNOSIS IN MALIGNANCY

As early lesions are brought to the surgeon with the question of malignancy the outlook for the cure of the patient becomes better while the difficulty of diagnosis is greatly increased. As the proper treatment of a malignant growth requires surgery to be of the most radical type, whereas benign growths can be easily eradicated by a conservative operation, the question of correct diagnosis is of prime importance. In order to be on the safe side every questionable lesion must be treated as malignant until proved otherwise.

When the surgeon saw cases of carcinoma of the breast, for example, presenting all the classical signs of malignancy, there was no difficulty in making the diagnosis and there was but little use in operating on the patient. Now when the surgeon is asked to diagnose a small freely movable lump in the breast of a middle-aged woman, it is practically impossible for him to know whether he is dealing with a malignant or non-malignant growth.

In such cases as these the value of an immediate frozen section is strikingly brought out. On the result of a pathological examination which can be performed in a very few minutes, while the patient is still under anesthesia, it is possible without prejudicing the welfare of the patient to perform a biopsy and so decide, as in the instance given above, between a simple excision of the growth or the amputation of the breast with the underlying muscles and the removal of the axillary contents. As Bloodgood* advises, "Biopsy must be done when a diagnosis of malignancy means an operation of greater magnitude than for a benign condition;

*Biopsy in Diagnosis of Malignancy. Southern Medical Journal, XX 1.

especially is this so when such a complete operation is mutilating or dangerous."

Of course, the most difficult part of this new demand on the surgeon is the technique of preparing the frozen section and its microscopic interpretation. There are few surgeons who have the time or inclination to work sufficiently with the microscope to render them familiar with this, perhaps the most difficult form of tissue diagnosis. It therefore seems that this field should be left to the trained pathologist, or at least to those surgeons who have had very considerable pathological experience.

The routine pathological examination of all surgical specimens is widespread and very valuable, but in the case of malignant diseases the ordinary type of pathological service is not sufficient. In fairness to the patient and operator alike immediate frozen section diagnosis by a competent man should be advised in every operation for malignant disease.

Bloodgood's opinion is that the best type of biopsy is one in which the tissue is exposed with thermal and chemical cauterization for protection against dissemination of tumor cells and the diagnosis is made from an immediate frozen section.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

CODMAN, ERNEST A., A.B. 1891; M.D. 1895 Harvard Medical School. His subject is "Obscure Lesions of the Shoulder; Rupture of the Supraspinatus Tendon." Address: 227 Beacon Street, Boston. Page 381.

ZINSSER, HANS, A.B., A.M., M.D., Columbia University. Former Professor of Bacteriology, Stanford University and College of Physicians and Surgeons, Columbia University. Professor of Bacteriology, Harvard Medical School. His subject is: "Hypersensitiveness." Address: Harvard University Medical School, Boston. Page 387.

LEECH, CLIFTON B., M.D. 1919 Boston University School of Medicine. Cardiologist, Union Hospital, Fall River General Hospital, Fall River; Member American Heart Association, New England Heart Association. His subject is: "Cactina Pillets." Address: 254 Rock Street, Fall River. Page 394.

BLACKFAN, KENNETH D., M.D. 1905 Albany. Professor of Pediatrics, Harvard Medical School. Address: Harvard Medical School, Boston. Associated with him

JOHNSTON, JOSEPH H., M.D. Assistant in Pediatrics, Harvard Medical School. Their subject is: "Notes on Infant Feeding." Address: Harvard Medical School, Boston. Page 395.

GUEST, GEORGE M., M.D. Assistant in Pediatrics, Harvard Medical School. His subject is: "Variations in the Diatase of the Blood of Infants." Address: Children's Hospital, Boston. Page 397.

CROTHERS, BRONSON, M.D. 1910 Harvard. Neurologist to Children's Hospital and Infants Hospital. Member American Pediatric Society, American Neurological Association; Instructor in Neurology and Pediatrics, Harvard Medical School. His subject is: "Obstetrical Injury of the Spinal Cord." Address: 270 Commonwealth Avenue, Boston. Page 397.

FULTON, JOHN B., B.Sc. 1921 Harvard, B.A. (Physiology) 1923 Oxford, Ph.D., M.A. 1925 Oxford. Demonstrator in Physiology, Oxford 1923-1925. His subject is: "The Early Phrenological Societies and Their Journals." Address: 126 Longwood Avenue, Boston. Page 398.

COUES, WILLIAM PEARCE, M.D. 1894 Harvard, F.A.C.S.; Surgeon to Out-Patient Department Massachusetts General Hospital. Assistant Surgeon, Boston Dispensary. Instructor in Surgery, Tufts College Medical School. His subject is: "The Spurzheim Collection of Phrenological Casts." Address: 9 Newbury Street, Boston. Page 400.

RECENT DEATHS

PRATT—DR. CHARLES ALBERT PRATT died at his home in Jamaica Plain, February 23, 1927, at the age of 76.

He was a graduate of the Eclectic Medical College of Maine, Lewiston, in 1885, and had practiced formerly in Chatham, Mass.

BULLARD—DR. JOHN THORNTON BULLARD died at New Bedford, of pneumonia, February 23, 1927, at the age of 62.

He was born in Boston March 31, 1864, his parents being John Lincoln and Sarah Walker (Spooner) Bullard. He studied at the Friends' Academy, New Bedford, and, entering Harvard, received his A.B. in 1884 and his M.D. from Harvard Medical School in 1887, after which he spent one year in Europe and another year in New York, beginning the practice of medicine and surgery in New Bedford, where he long continued in active practice.

In 1890 he was appointed on the surgical staff of St. Luke's Hospital, New Bedford, and for a long time was one of the visiting surgeons. In 1896 Dr. Bullard was appointed a member of the Board of Health of New Bedford, a post he held for eight years. In 1895 he was appointed associate medical examiner for Bristol County. From 1889 to 1919 he was a Fellow of the Massachusetts Medical Society.

He was a director of the Rotch Spinning Company before its absorption into the New England Cotton Yarn Company, and for some time was a director of the Potomaska Mills, the Pierce Manufacturing Company and the Fairpoint Corporation. He was also a trustee of Friends' Academy. He helped organize the Country Club of New Bedford and was its president for the first five years. He was a member of the Somerset Club of Boston and numerous medical bodies, and was president of the Harvard Club of New Bedford.

On June 18, 1889, Dr. Bullard married, at New Bedford, Mass., Miss Emily Morgan Rotch, daughter of William J. and Emily (Morgan) Rotch. They had five children.

CORRESPONDENCE

TISSUE DIAGNOSIS IN THE OPERATING ROOM AND IMMEDIATE COVER-SLIP EXAMINATIONS OF ALL FLUIDS AND PUS

Baltimore, February 3, 1927.

Dear Sir:

I will consider it a courtesy if you will publish this letter in your journal, as I am anxious to come in correspondence with pathologists and surgeons interested in the immediate examination, by frozen section, of tissue in the operating room and the immediate cover-slip studies of smears from all fluids and pus.

Microscopic examination of stained frozen sections has been possible for more than a quarter of a century. The staining of unfixed frozen sections with polychrome methylene blue and other stains is a well-established procedure. In many operating rooms in university and other large and small surgical clinics, provisions for these immediate diagnostic studies have not only been available, but have been in practical use for years. While, unfortunately, on the other side, this diagnostic part of the operating room is conspicuous by its absence in many clinics.

Before 1915 it was rarely necessary for a surgeon well trained in gross pathology to need a frozen section to help him in diagnosis at the operating table. Since 1915, and especially since 1922, the public has become so enlightened that malignant disease formerly easily recognized either clinically or in the gross, now appears in our operating rooms devoid of its easily recognized clinical and gross appearance and can only be properly discovered by an immediate frozen section. The majority of operating rooms are not equipped or prepared for this new diagnostic test.

The first essential part for this diagnosis is the technician—one to cut and stain the frozen section, or to make and stain the smear. The second is a pathologist trained to interpret it. It is possible for the surgeon to be all three in himself, and some young surgeons are so equipped. In others it is a dual combination—surgeon and pathologist in one, and the technician. More frequently it is three—operator, technician and pathologist. It makes little difference whether it is one, two or three individuals, providing each has the equipment and training for this most difficult diagnostic test.

In the address as chairman of the Surgical Section of the Southern Medical Association, I discussed biopsy, and this paper has been published in the *Southern Medical Journal* for January, 1927 (Vol. XX, page 18). A reprint of this paper will be sent to anyone on request. The chief object of this letter is to come in contact with surgeons and pathologists who are sufficiently interested in this problem to discuss it either by correspondence, or by attending a meeting in the surgical pathological laboratory of the Johns Hopkins Hospital, either the Monday before, or the Friday after the meeting of the American Medical Association in Washington.

Schools for technicians may have to be established in different sections of the country, and the surgical pathological laboratories of the medical schools and the larger surgical clinics should offer courses in this tissue diagnosis, so that surgeons may learn to become their own pathologists, or pathologists learn the particular needs of the surgeon in tissue diagnosis in the operating room.

It is quite true that when the majority of the pub-

lic are fully enlightened, the surgeon will see lesions of the skin and oral cavity and the majority of subcutaneous tumors when they are so small that their complete excision is not only indicated, but possible without any mutilation. The chief danger here will be a surgical mistake—the incomplete removal of an apparently innocent tumor. There is no necessity here for biopsy. If a proper local excision is done, no matter what the microscope reveals, that local operation should be sufficient. But when lesions of the skin, oral cavity and soft parts are extensive and their complete radical removal mutilating, then there must be biopsy to establish the exact pathology.

In tumors of the breast and disease of bone, for years, the diagnosis could be made clinically, or from the gross appearances at exploration. But now, an increasing number of cases, the breast tumor must be explored, and the gross pathology of this earlier stage is not sufficiently differentiated to allow a positive diagnosis. Immediate frozen sections are essential to indicate when the complete operation should be done. The same is true of the earlier stages of lesions of bone. The X-rays no longer make a positive differentiation between many of the benign and malignant diseases, for example, sclerosing osteomyelitis and sclerosing osteosarcoma.

We must not only specialize in tissue diagnosis, but we must organize this department so it will function properly in as many operating rooms as possible in this country.

Then there is a final and most difficult question to consider. I doubt if it can be settled. What shall be done in those operating rooms in which there is no technician to make the sections and no one trained to interpret the microscopic picture? How can a piece be excised or a tumor removed, for example, from the breast, and this tissue sent to some laboratory for diagnosis without incurring the risk of the delay to the patient? I have discussed this point in my paper on biopsy.

JOSEPH COLT BLOODGOOD.

Surgical Pathological Laboratory,
Johns Hopkins Hospital.

NOTICE

EDWARD K. DUNHAM LECTURES

THE Faculty of Medicine of Harvard University announces that two lectures are to be given under the Edward K. Dunham Lectureship for the Promotion of the Medical Sciences on "Progress in Enzyme Research." Tuesday, March 29, "Some Recent Problems." Wednesday, March 30, "Methods and Results." At five o'clock at the Harvard Medical School Amphitheatre, Building C, by Richard Willstätter, Ph.D., M.D. (hon.), Nobel Laureate, Privy Councillor, and Professor in the University of Munich.

THE EDWARD K. DUNHAM LECTURESHIP

In 1923 there was founded in memory of Doctor Edward K. Dunham (M.D., Harvard, 1886), the Edward K. Dunham Lectureship for the Promotion of the Medical Sciences. Among the useful purposes for which the Foundation was established was that of binding closer "the bonds of fellowship and understanding between students and investigators in this and foreign countries." The lecturers are chosen from

"eminent investigators and teachers in one of the branches of the Medical Sciences, or of the basic Sciences which contribute towards the advance of Medical Science in the broadest sense." The lectures, which are given annually, are "free and open to the faculty and students of the Harvard Medical School and College, and all other interested professional persons who may profit by them."

REPORTS AND NOTICES OF MEETINGS

GRADUATE COURSES IN PEDIATRICS

COURSE A. A one-month all-day course offered in June. Lectures, clinics, case teaching, food demonstrations, and other exercises.

Fee: all day, \$100; mornings only, \$60; afternoons only \$40; afternoons with lectures, \$60.

COURSE B. A course of six to twelve months of general pediatric training. Further particulars on application.

Fee: per month, \$50.

For information apply to Secretary, Courses for Graduates, Harvard Medical School, 240 Longwood Avenue, Boston, Mass.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

THE Essex South District Medical Society held its regular meeting at the Lynn Hospital, Wednesday, March 2, 1927.

Clinic by members of the Staff at 5 P. M.

Dinner at 7 P. M.

Dr. George R. Minot of Boston spoke on "Pernicious Anemia With Special Reference to Liver Diet."

Discussion by Dr. A. N. Sargent of Salem and Dr. E. D. Reynolds of Danvers, 10 minutes each. General discussion from the floor.

Attendance 75.

WM. T. HOPKINS, *Reporter*.

BOSTON MEDICAL HISTORY CLUB

A MEETING of the Club was held at the Warren Museum, Harvard Medical School, January 28th, 1927.

The first paper of the evening was by Dr. Robey on "Dr. Robert Knox and the Edinburgh Murders. This paper will appear in a succeeding issue of the JOURNAL.

The rest of the meeting was given over to the subject of Phrenology, based on the collection of casts at the Warren Museum. Papers on this subject were read by Dr. Cones and Dr. Fulton. Both papers appear in this issue of the JOURNAL.

Dr. Harvey Cushing spoke briefly on the place that Spurzheim and Gall should take in the history of medicine. He felt that they were important in breaking down the old Galenic idea of vital spirits as connected with the ventricles of the brain and that they prepared the way for,

and made the medical profession ready to accept, the more definite determination of cerebral localization as developed by Broca, Ferrier and others later in the nineteenth century. Dr. Cushing then presented a large oil portrait of Spurzheim to the Warren Museum. This portrait was formerly the property of Dr. J. C. Warren and was painted shortly after Spurzheim's death, in Boston, in 1832. It is a very striking portrait, excellently preserved.

WEST END NEIGHBORHOOD CONFERENCE

A MEETING of the West End Neighborhood Conference will be held at the Blossom Street Health Unit Friday, March 11, 1927, at 3:45 P. M.

Dr. Charles F. Wilinsky, Deputy Health Commissioner will speak on the "Public Health Program of Boston." You are cordially invited to be present and bring friends.

THE NORFOLK DISTRICT MEDICAL SOCIETY

A REGULAR meeting of the Society was held in the Roxbury Masonic Temple, 171 Warren Street, Roxbury, March 1st, 1927, at 8.15 P. M.

Dr. R. B. Greenough reviewed the cancer question and discussed the modern methods of handling this problem. At the close of his paper a general discussion followed.

SOCIETY MEETINGS

DISTRICT MEDICAL SOCIETIES

Essex North District Medical Society

Wednesday, May 4, 1927—Annual meeting. Russell Hall, Young Men's Christian Association Building, 40 Lawrence Street, Lawrence.

Thursday, May 5, 1927—Censors meet for examination of candidates at Hotel Bartlett, 95 Main Street, Haverhill, at 2 P. M.

Essex South District Medical Society

Wednesday, April 6, 1927—Danvers State Hospital. Clinic, 5 P. M. Dr. Allan W. Rowe, Chief of Research Service at Evans Memorial, "The Differential Diagnosis of Endocrine Disorders." Followed by dinner. Discussion by Drs. Wood of Hathorne and Kline of Beverly, ten minutes each.

Thursday, May 5, 1927—Censors meet for examination of candidates at the Salem Hospital, 3:30 P. M.

Wednesday, May 11, 1927—Annual meeting. The Tavern, Gloucester. Speaker and subject to be announced later.

Norfolk District Medical Society

Below are the proposed meetings of the Norfolk District for the remainder of the year. Minor changes may be made in case of necessity.

March 29, 1927—Roxbury Masonic Temple, 8:15 P. M. Drs. F. S. Newell and F. J. Irving, "The Modern Treatment of the Eclampsias and Toxaemias of Pregnancy." If time permits—"The Modern Methods of Handling Prospective Caesarean Cases."

May 10, 1927—Annual meeting. Details of meeting to be announced.

Suffolk District Medical Society

Meetings of the Suffolk District Medical Society and the Boston Medical Library will be held at the Boston Medical Library, The Fenway, Boston, at 8:15 P. M., as follows:

March 30, 1927—Medical Section. Subject and speaker to be announced later.

April 27, 1927—Annual meeting. Election of officers. "Medical Education in the Orient and Occident," Dr. David L. Edsall, Dean, Harvard Medical School.

Notices of meetings must reach the JOURNAL office on the Friday preceding the date of issue in which they are to appear.